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THE PRESENT STATUS OF OTITIC MENINGITIS.*

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I feel almost like apologizing for bringing up the question of meningitis again this year, especially when two years ago at the American Otological Society meeting we had a symposium at Atlantic City, and last year the question came up when Dr. Kopetsky took up some special aspects of the chemistry of the spinal fluid. Again at the meeting in Toronto, as chairman of the committee on meningitis, we reported on the work we had done in the past year. However, I do not apologize at all when it comes to the question of going from hospital to hospital in New York City and seeing cases of meningitis in which the diagnosis is not at all clear. It is not at all clear in another sense of the word because we fail to realize, as rhinologists and otologists, the fundamental basis on which a diagnosis of acute purulent, that is, generalized meningitis should be made. There is less apology necessary, too, when you try to reconcile the literature; when you try to reconcile what a man reports as 40 or 50 or 60 per cent of cures of acute purulent meningitis with what other men have found; myself, for instance. In 28 years I have seen 367 cases of meningitis with 18 recoveries, and why they recovered I do not know. I have analyzed the cases which recovered, and I do not believe it was anything we did that cured them. I propose to go into the clinical pathology of meningitis from the standpoint of diagnosis because that is where the literature falls down in such cases

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as this. Two years ago when I was preparing my part of the symposium at Atlantic City, in going over the literature I saw such cases as this: "Spinal fluid cloudy; case cured." It is to clear up these things that I speak.

I propose to speak on three different points: The examination of the spinal fluid; a very important point that is not generally recognized, the blood count; and the pathways of infection.

First of all, we must agree on what acute purulent meningitis is. The New York Committee of the American Otological Society formulated a very definite definition, that we must demonstrate the organisms, either by smear or culture or both, in the spinal fluid before we can consider a case as acute purulent meningitis. I fully realize that an odd case, a very, very rare case, may give us a matting of pus all over the brain and still the spinal fluid be clear, but I have only heard of one case, in all the literature of this kind, of which I am definitely sure.

In the examination of the spinal fluid, first, we go into the question of its physical characteristics; second, the chemistry; third, the cytology; fourth, the serology; and fifth, the bacteriological characteristics. These are the things we keep in mind in the examination of the spinal fluid. I am not going into the chemical characteristics because that was dealt with very exhaustively last year. The physical characteristics of the spinal fluid in meningitis are two: Either it is clear or not clear. If it is a clear spinal fluid and it stays clear, and we have the symptoms of meningitis, it can be only one of two forms, either tuberculous or syphilitic; if it is tuberculous meningitis, by long centrifuging of the fluid we can usually demonstrate the tubercle bacillus. If it is the syphilitic form we are usually aided by the cytology, the cell count being lymphocytes and, of course, the serology a + Wassermann reaction. So much for the physical characteristics.

The next point to consider is a cloudy fluid, and here is where all the trouble arises in the diagnosis of meningitis. A number of the reports in the literature base the diagnosis of generalized meningitis on the fact that the fluid is cloudy, in spite of the fact that organisms are not demonstrable. A cloudy fluid in itself, to my mind, does not mean generalized meningitis. We get many cloudy fluids with symptoms of meningitis and no organisms. We lose sight of a fact which we see all

the time in eye work. In the anterior chamber of the eye there is a clear fluid corresponding to the spinal fluid. If there is an infection of the cornea, such as a pneumococcic or streptococcic infection, the anterior chamber is filled with pus. This is called hypopyon. The fluid is sterile, with no organisms in it. The explanation is that this extracorneal or extra-anterior chamber infection has kicked up enough reaction in the tissues to cause a transudation of the leukocytes from the iris or ciliary body, and we get a sterile effusion of pus. My point is that in all of these cases with a cloudy spinal fluid with no organisms, there is an extradural infection. We get this time and again in an extradural abscess and time and again where a sequestrum of bone has formed: It is irritating the external layers of the dura but has not penetrated the dura. To illustrate that point, last winter Dr. Blackwell reported one of the most interesting cases to bear out this point. I do not know whether he reported it before this Section or not. The patient was operated on at his clinic four different times for a mastoid. The patient had a cloudy spinal fluid for 32 days, but never at any time were organisms found. Finally, when Dr. Blackwell operated he found a sequestrum of the petrous process of the mastoid bone. In two days the spinal fluid was clear. Here was nature throwing out a tremendous response to try to get rid of this sequestrum. On our own service last winter we had a man with symptoms of meningitis. We decided that as long as the blood count was up it was unwise to go in and search for a cerebellar abscess. The canal of the ear was pouring out pus. He developed complete facial paralysis and finally, as he got worse, we prepared to go after a cerebellar abscess. When we operated we found a bone sequestrum as in Dr. Blackwell's case, and the spinal fluid was negative within 36 hours, although he had had an increased cloudiness of the fluid for over two weeks before we operated. There was a case of infection outside the dura giving this reaction. What first drew my attention to this, years ago, was a patient admitted to the hospital with a blood count of 30,000 leukocytes and a temperature of 106. He had a tremendous reaction and had to be tied down when the ambulance brought him in because of his raving. He had every symptom of meningitis. When I did a lumbar puncture the fluid was under such pressure that it shot across to the next bed; this was at night. The next morning his ears, nose and throat were negative; he cleared up and never had any more trouble. He had a profound protein intoxi-

cation. These are the cases we miss out on; these are the cases which are reported in the literature as cured cases of purulent meningitis. To my mind they are extradural localized meningitis and not the type we are speaking of tonight.

Nothing need be said about the cell count and the serology I want to dwell on the bacteriological aspects of the spinal fluid. This is where another great contradiction occurs in our conceptions, because if we have no knowledge of the bacteriology of the spinal fluid we cannot get on. The literature is full of cases in which the organisms can be demonstrated in smears but cannot be grown on culture. Where they get two or three organisms in the same smear staining differently, they report two organisms present. We must get this firmly fixed in our minds. In my experience two organisms in meningitis is a very rare finding. It may occur as a terminal thing where, for instance, a meningitis has been caused by the streptococcus or pneumococcus and the patient has been ill for months; and, rarely, there may occur a second infection with B. coli. We must remember that the spinal fluid has bacteriocidal properties and if an organism is present for some time, it may become attenuated and lose its staining properties. This is important because of the fact that most of the organisms we see in the spinal fluid are either gram-positive or gram-negative. The gram-positive organisms are the streptococcus, the pneumococcus (there are 32 different varieties of these,) and the staphylococcus. The gram-negative is the meningococcus or the epidemic type. The gram-positive organisms stain violet with the gram stain, but what is not well realized is the fact that if these organisms, which are normally gram-positive, are injured or undergo autolysis, they lose this property of taking the gram stain and become gram-negative. They are then looked upon as a mixed culture of pneumococcus or streptococcus and the meningococcus. Hence, in many cases when these mixed cultures are found, they mistakenly give the patients antimeningococcus serum and treat the case as one of epidemic meningitis when all the time it is a case of an ear infection. In the *Journal American Medical Association* a case was reported last spring that bears out this point excellently: The patient had symptoms of meningitis with a cloudy fluid. On the first smears they found a gram-positive and a gram-negative organism. They immediately gave him antimeningococcus serum. The next day after culturing the

fluid they got what they thought was a streptococcus and immediately injected antistreptococcus serum. Two days later the organism was identified and the patient was found to be suffering from a staphylococcus infection from which he recovered. It has been my experience that better response to treatment is obtained in meningitis due to injury. This explains, therefore, why we get these different organisms. Another point is, if you have a patient with a running ear and meningitis, and you come up against this problem of a gram-positive and a gram-negative organism, give him the benefit of the doubt that it is not epidemic meningitis but an ear case.

The next point in the problem of meningitis is this: The question was asked me last year whether the presence of an organism in the spinal fluid means meningitis in every case. Probably in 99 per cent of the cases it does, but here is a case I saw in Boston, with Dr. Crocker. The patient was a woman, age 32 years, operated on for radical mastoid by the house surgeon. She never awoke from the anesthetic but continued with normal breathing and a normal pulse. She had no symptoms of meningitis but was simply in coma. A lumbar puncture was made and the spinal fluid was cloudy and loaded with organisms which stained gram-positive and gram-negative. While the patient was unconscious, diagnosis of a ruptured brain abscess was made, and she got well. They were able to demonstrate the empty brain abscess cavity. The organisms in the pus had been so injured that they could not be grown in cultures; culture after culture was made but no organisms grew. The cases in which the organisms are demonstrated in the smear but not in the culture are due to this same explanation. The organisms undergo degeneration due to the serum. The thing to do, therefore, is to plant not a loopful of spinal fluid but one or two cubic centimeters. I remember two cases in which the cultures were negative, and we planted big streaks of pus on the plates and all we got were 10 colonies of the pneumococcus.

A very important point to bring up is the question of the blood count. For years and years it has been my policy in nose, throat and ear cases where there is a high leukocyte count, that is, over 25,000 to 30,000, with a high polymorphonuclear count (except in children), to say that it is a brain abscess or it is pneumonia. This has helped me out time and again in getting after these cases early. A few years ago we

had a very unfortunate experience with one of Dr. Duel's cases: We followed this case from the standpoint of the clinical pathology for over six weeks. The patient was out driving his car after a mastoid, but never in the six weeks did he have less than 42,000 leukocytes. The count ran from 42,000 to 53,000 leukocytes and the polynuclears were never under 93 to 94 per cent. Although he seemed just as normal as you and I, we knew there was something going on, which later proved to be a staphylococcus pyogenes infection. This organism is looked upon as a nonpathogenic organism. The key to this case was the high blood count. Although the patient was eating normally and was out driving, we knew that he was doomed. Two weeks after the mastoid he developed a swelling in the neck and finally developed an acute cerebellar abscess. We operated but it was not localized. We would never have been justified in invading his meninges without that high blood count. That is a very important point, which has helped me personally to decide when to go in on a brain abscess. One is not justified in going in if the blood count is high because the abscess is not yet localized; there is only a cerebritis. I reported this case at the American Otological Society.

Another point in the handling of these cases is the question of the pathway of infection. We must change our conception of these cases. I had always thought that the infection traveled in from the mastoid. I have come to the conclusion that a large majority of our mastoid deaths are really not due to the mastoid. In other words, there is an extension from the sphenoid process, probably in the form of a low grade osteomyelitis or a low grade thrombophlebitis, which causes the meningitis, rather than extension from the mastoid or mastoid surgery in itself. Two cases in which I did not have the opportunity of doing a complete autopsy but got the bones out after death, proved that point to my satisfaction. One patient had mastoiditis, which was treated conservatively by one of the best men in New York; four weeks later he died of meningitis. The base of the mastoid bone and the whole canal was removed, and the bone was found to be infiltrated with pus. In other words, we operated on the mastoid portion, but we could not operate on the rest of the bone; that would have been impossible. Some of these are osteomyelitic cases, I believe, from the very moment the infection occurs, and they are lost from that day on, no matter what we do. That brings up the

question as to whether we are justified in doing a mastoid in a case of meningitis with a running ear. I have revised my opinions very much about this point in view of some of the cases I have seen under the auspices of the American Otological Society. One case from Dr. Duel's service had had a pain in his ear on Monday and by Wednesday night was brought in to us raving with meningitis. That night a myringotomy was done by the house surgeon, and, fortunately, a culture was made which was negative. Several of us, including Dr. Duel, Dr. Tickle and Dr. Hare, went over him very carefully, and we came to the conclusion that although he had a running ear on the left side, it was not a mastoid case. He had some history of sinus trouble. We decided not to do a mastoid operation on him, and I am very glad that we didn't. We got permission after death to take out the mastoid bone and there was no doubt that it was a sphenoid infection and not a mastoid. We must keep in mind that a lot of these infections travel in through the lymphoid tissue of the nasopharynx and are not necessarily caused by our surgery.

In conclusion, here is a case I want to cite to illustrate what is happening and where we, as otologists, ought to be more on our toes. This case was admitted to the medical service of the hospital with all the classical symptoms of meningitis. Patient was kept there for two days and the ear men were not notified, in spite of the fact that the ear was running. He had a leukocyte count of 18,000 with 84 per cent polynuclears. For two days a tentative diagnosis was made of tuberculous meningitis. Lumbar puncture was done and the fluid was clear. They lost track of the fact that he would not have had a leukocytosis if this were a tuberculous meningitis. He was then transferred to the otological service. At operation a rotten mastoid was found, and he died two days later.

These practical points which we are up against are cited so that they may help us to make a clear diagnosis early. We must keep in mind these objectives and the information that may be revealed by the microscope and the blood count.

57 East 57th Street.

FRACTURE OF BASE OF SKULL AND OTOSCLEROSIS.*

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The interesting coincidence of fracture of the base of the skull and of otosclerosis itself, as well as the more recent considerations pointing to some relationship between fracture and bone fragility respectively, and otosclerosis seem to warrant publication of the following case.

REPORT OF CASE.

The patient was a shoemaker, age 57 years, father of four healthy children. Except for measles in childhood he never was seriously ill. His hearing had been impaired for a long time. Feb. 18, while intoxicated, he was struck by a tramway and fell, striking the back of his head; he did not lose consciousness.

On admission, patient's breath had an alcoholic smell, and he complained of dizziness. On the top of the head and just above the right tuberfrontale there were bruises covered with crusts. The skin surrounding the right eye showed a bluish discoloration. Left facial palsy, by which the nasolabial fold was flattened out. Temperature, 37.5° C.; no pressure fluid was observed. There was a discharge of a clear fluid from the left ear, at times mixed with some blood.

Otologic Findings: Right ear shows no pathologic change either externally or otoscopically. From the left external auditory canal blood clots were carefully removed with the aid of a forceps; after this there was free discharge of bloody cerebrospinal fluid. About the junction of the cartilaginous and bony portions of the auditory canal, on the upper-posterior wall, was a wound of the lining skin, 3 cm. long, with some bleeding. At this site the auditory canal was narrow and but a partial view of the membrana tympani could be had. From this region there was an intermittent pulsating discharge of cerebrospinal fluid, tinged with blood. The visi-

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ble part of the membrana, the lower half, showed no tear. The skin of the ear and of its close neighborhood was normal. There was a tenderness on pressure of the mastoid process and of the temporomandibular joint. Complete left-sided facial palsy. Lumbar puncture yielded clear cerebrospinal fluid.

Lateral Roentgenogram showed a fissure, 3 cm. long, extending from the parietomastoidal suture into the petrous bone. It is shown also by the X-ray picture taken according to the method of Mayer; here it takes its course at the junction of the pars tympanica and pars petrosa, perpendicularly to the axis of the pyramid, with an irregular serrate line down to the anterior margin of the petrous bone.

Functional Examination: There were no spontaneous labyrinth symptoms, except for feeling of dizziness. Data of value concerning hearing acuity are as follows: On the right side loud conversational tone was perceived; on the left side only shouting was heard. Tuning forks: C_1 was perceived as well from the crown of the head as from the occipital bone; by aerial conduction. Right, only C_5; left, all tuning forks from C_3 upward.

Course of the Illness:. Feb. 20, sudden change for the worse, restlessness, no loss of consciousness. Pulse rate, 132. Feb. 21, great decrease of discharge of cerebrospinal fluid from the ear, bloody tinge almost disappeared. Feb. 22, death occurred, with signs of progressive cardiac failure.

Autopsy: On the left side in the posterior parietal region and in the midpart of the parietal bone, as well as in the occipital region, there were breaks of the skin, which at the sites mentioned showed a pale livid discoloration. After removal of the calvarium the meninges were seen to be extensively covered in this region and at several other sites all over the convexity by a fibrinopurulent layer; there were many blood clots between meninges and brain and covering the meninges. After stripping off the dura on the anterior surface of the left petrous bone, a bone fissure was visible, which at the junction of the lateral and middle thirds of the pyramid passed over upon the posterior aspect of the same and could be followed along the sigmoid sulcus in the immediate proximity of the lambdoid suture up to the level where the calvarium has been sawed off, its total length being

13 cm. By a branching off, a piece measuring 8 x 9 cm., corresponding to the tympanic antrum, was broken out from the bony wall of the pyramid; this piece, however, was held in place by the adjacent structures. There are no other changes on the base of the skull. Cause of death: Fibrinopurulent meningitis caused by infection subsequent to blunt traumatism to the left retroauricular region with fracture of the base of the skull.

Extrinsic causes permitted us to remove and to work up only the left (the injured) petrous bone. This was cut into a series of 655 vertical sections. Microscopic examination yielded the following results:

Fig. 1. General view, vertical section. Fracture of external auditory meatus with prolapse of soft parts, tear of the flaccid membrane, entrance of blood and desquamated epithelia from the external auditory meatus into the tympanic antrum, penetrating fracture of the tegmen, severance of the nerve in the internal meatus.

In general there was a distinct pneumatic structure. In the external auditory canal the desquamated epithelium was embedded into blood clots; on the back wall a fracture was seen extending along the entire section series, from this fracture cellular masses were herniating. The tissue of the membrana tympani was hyperemic, and masses of clotted blood adhered to both sides. There was a perforation in the pars flaccida, which, like the fracture, followed along the entire series of sections. The tear began at the center of the well developed papillary border zone; the part of the membrana attached to it was retracted superiorly-posteriorly and covered by heavy masses of blood clots. Through the rent, clots containing scaled off epithelia protruded into the tympanic cavity. In the tympanic cavity the fracture immedi-

ately attracts attention; it crossed the tegmen tympani, thus exposing the subdural area over the attic. The medullary cavities of the abundantly pneumatic tegmen contained distended blood vessels and also extravasations. This fracture of the tegmen may be followed through the entire series of sections in the same way as that of the back wall of the auditory meatus and the break of Shrapnell's membrane. In the lumen of the tympanic antrum no pathologic content was found; there was some blood only in the recesses, as between the crura of the stapes; the entire hypotympanum was free. The ossicular chain was dislocated from its original site.

The bony labyrinth was not injured by the traumatism. No break of continuity was found. There was an otosclerotic focus of typical structure and location, at the site of election, adjacent to and surrounding the footplate of the stapes. The latter was incorporated in this focus in its entirety; from here the pathologic bone extended some distance into the capsule of the labyrinth to both sides, against the promontorium as well as the facial canal. The focus may be followed in the vertical series from section No. 215 till No. 390. Not only was the footplate of the stapes completely pent up, but the focus at the promontorium, which occupies the entire wall of the niche of the oval window at the opening of the narrow and deep recess was fused with the inferior crus of the stapes. In this way both one crus and footplate of the stapes were firmly adhered to the labyrinth wall. The otosclerotic bone showed the characteristic structure and was of a sclerotic, amedullary type. Its line of separation against the healthy bone was, as usually, quite sharp. The medullary cavities were partly filled by distended blood vessels. At a point near to the facial canal the sharp line of boundary was crossed by a distended blood vessel, which could be followed some distance in both directions.

The membrana tympani secundaria showed extravasations towards both middle and inner ear. There were no remarkable changes in the cavities of the inner ear. There were no free blood collections either in the endolymphatic or in the perilymphatic space. Attention was attracted only by a considerable hemorrhage in the stria vascularis, which was most extensive in the middle convolution. Here it was visible from section No. 100 to No. 150; traces, however, were found also in the stria of the basal convolution, whereas the top convo-

lution was free. Reissner's membrane was pressed to the papilla or glued by blood to the strial wall, respectively, in the basal convolution; in the other coils it was tense. In the internal auditory canal perineural hemorrhages surrounded the trunk of the auditory nerve.

Summary of the Facts: A man, age 57 years, who, struck by a tramway, sustained a fracture of the base of the skull, showed with relatively insignificant external wounds a bloody serous pulsating discharge from the left ear (cerebrospinal fluid), which after 24 hours became minimal; X-ray picture showed a fracture of the parietal bone and of the pars tympanica. There was a great impairment of hearing, only the high-pitched tuning forks being perceived. Complete left-

Fig. 2. Part of preceding Fig. 1, under higher magnification. Fracture of the bony wall of the external auditory meatus with prolapse, tear of Shrapnell's membrane sharp at the papillary border zone, with intruding blood clots and desquamated epithelia.

sided facial palsy. Death occurred on the fourth day. Post-mortem showed wounds of the soft parts of the left parietal and occipital region, fibrinopurulent meningitis, subdural hemorrhages and a bone fissure of the pyramid. A series of histologic sections made from the left petrous bone exhibited a fracture of the back wall of the external auditory meatus and of the tegmen tympani, a tear of the drumhead in the region of the flaccid membrane. There were considerable amounts of blood in the external, and smaller amounts in the internal auditory meatus and in the niches of the tympanic cavity; hemorrhages in some parts of the cochlea; also a typical otosclerotic focus was found, which completely encom-

passed the footplate of the stapes and was fused also with one of the crura.

COMMENTS.

Reflections provoked by the course of this case concern, in the first place, the injury itself.

First, we should like to call attention to desquamated epithelia of the external auditory canal, which, embedded into blood clots, was forced into the tympanic antrum. We had the occasion (*Acta Oto.-Laryng.*, 20:211, 1934) to comment on the process which, on a traumatic basis, may give rise to the development of a cholesteatoma. In this case all the conditions were fulfilled, which, in the case of recovery, without consecutive meningitis, could positively lead to a late cholesteatoma: tear of Shrapnell's membrane, rolling up of the edges of the perforation as a basis of cyst formation, intrusion of epithelial products, the latter being the origin of the inclusion of a later cholesteatomatous formation; also a stimulus for further epithelial proliferation and for secondary inflammatory conditions, and so on. Of course, on account of the rapid fatal issue in the present case there was no possibility of development into this direction.

As to the quality of osseous tissue of the petrous bone, there was a good pneumatization, also the fractured tegmen consisted of several rows of well developed cells.

Among preformed formations the fissures often found in the osseous tissue of the capsule of the inner ear should receive mention in the negative sense, however, to stress their absence. Only at two sites could such fissures be detected: one originating from a semicircular canal; the other, taking its course across the capsule of the cochlea in the prolongation of the lamina spiralis at the level of the middle coil. Both exhibited strands of connective tissue within their lumina, as an evidence of their existence preceding the lethal traumatism. This finding was of two-fold importance: In the first place it can be stated that the fracture had no connection whatever with the genesis of the fissures, nor did it cause any microscopic breaks; secondly, the theory insisting on a relation between otosclerosis and these fissures (O. Mayer) may be mentioned. Without wishing to draw further conclusions from this case as to the item mentioned, it should be empha-

sized that the otosclerotic focus developed in a petrous bone almost devoid of microfissures of typical appearance and sites. In this way no direct relationship could be demonstrated between fracture and otosclerosis, as stated in my previous paper (*Zeitschrift. f. Hals.-Nasen u. Ohrenheilk.*, 23:588, 1934); nor any coincidence with a general bone fragility, no signs of which were observed.

Concerning comparison of diagnosis of fracture in the living patient and by X-ray examination with data of autopsy and microscopic examination, a by no way rare occurrence should be noted: a break of continuity, which by the X-ray picture is shown to run perpendicularly to the axis of the pyramid, proved not to extend into the inner ear. The process

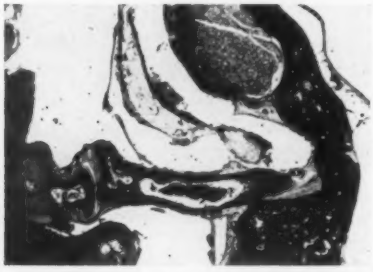

Fig. 3. Otosclerotically pent up footplate of the stapes, otosclerotic focus fused once more with crus of stapes at promontorium.

may be clearly reconstructed by the aid of a series of sections from the region of the promontorium: the fracture running across the tegmen tympani does not penetrate into the capsule of the cochlea; if a force from above is applied, the same is abducted along the medial wall of the tympanic antrum. It glides off, so to say, on the surface of the promontorium and it is a question as to whether or not the otosclerotic focus acted as a reinforcement. Also, H. Richter (*A. Ohrenheilk.*, 139:181, 1935), in his case of fatal meningitis subsequent to operative luxation of an otosclerotic stapes, was unable to decide whether otosclerotic changes facilitate dislocation, or, to the contrary, by thickening entail an increased resistance to traumatism. Correspondingly to tissue destruction behind and above the ear, this

region must be considered as point of departure of traumatism in the sense that the patient had been knocked down, hitting the back of his head against the pavement; from here the fracture line took its origin, branching off at the medial (anterior) surface of the pyramid, and encircled the inner ear like a claw, similar to the grasp of bent fingers of the hand around a ball.

The fracture of the back wall of the auditory meatus could be viewed otoscopically; microscopic examination but verified clinical findings.

The relative intactness of the inner ear amidst its injured surroundings was evidenced by tests of hearing; however,

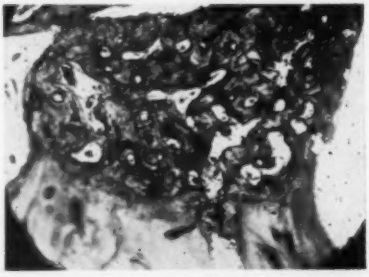

Fig. 4. Otosclerotic focus of promontorium, together with site of fusion with crus of the stirrup; sharp limitation against the healthy bone.

it could be wholly understood only after histologic examination. Tuning forks were perceived in the examined (left) ear from C_3 upward, as mentioned in the case history. On the basis of findings in the three convolutions this seems to be paradoxical, strial hemorrhages being found in the basal and middle coils, only the top coil entirely free, thus leaving a part destined for the reception of low tones; this finding, however, cannot be considered as an implicit effect of traumatism. It is to be borne in mind that O. Mayer (1917), found hyperemia and swelling of the vascular stria in otosclerosis. Hearing, however, is not determined by changes in the cavities and soft parts of the cochlea mentioned but rather by the otosclerotic focus. The upward shift of the lower tone perception limit is attributable to otosclerosis.

The incongruence between functional and microscopic cochlear findings is another evidence of the independence of impairment of hearing from the injury, as well as from bleedings in the internal auditory meatus. The impairment of hearing is simply a characteristic loss of function in otosclerosis. In this way the inner ear suffered no functional injury from the traumatism. The fact that in the right ear, which was not examined histologically, tuning forks were heard only from C_3 upward in all probability indicates that both ears were affected by the otosclerotic process, as is the general rule.

One question is still to be answered: whether immediate operative intervention could not have offered better chances

Fig. 5. Bleeding in vascular stria of middle convolution.

for recovery than delay in this procedure? Certainly, the discharge of cerebrospinal fluid warranted exposing the break through which it escaped, thus controlling any ascending infection; however, multiplicity of injuries dissuaded us from an intervention at this single site. Meningitis, which possibly originated at several points simultaneously from scattered hemorrhages, also proved that fatal termination of the process could not have been prevented by any intervention on the petrous bone.

This case demonstrates how clinical findings were elucidated by the X-ray picture; how, in turn, the suggestions of the latter were verified by autopsy and microscopic examination, respectively, and how hearing tests, too, received adequate interpretation by the data of the latter. Only complete

microscopic working up of the case could corroborate inferences drawn from clinical data. However cogent the latter were, they could not be sufficiently supported by data of the postmortem. Among others, I wish to point to the fact that without microscopic examination medicolegal evaluation of the case would have been imperfect.

SUMMARY.

Traumatism by being struck by a tramway, consecutive fracture of the base of the skull with involvement of the petrous bone. Death of the patient on the fourth day, due to meningitis. Anatomical and functional findings made on the living patient could be correctly interpreted only after careful examination of a series of microscopic sections of the injured petrous bone. The sections showed also an otosclerotic focus. It has been proven that the inner ear has remained intact in the center of a seriously injured domain and that impairment of hearing was due to pre-existent otosclerosis.

Reáltanoda-u.9.

The First International Congress of Sanatoria and Private Nursing Homes will be held in Budapest at the end of September next. Special invitations shall be sent in due course to all parties concerned. The short extract of lectures, discussions, proposals, etc., should be sent to the following address as early as possible in order that they may be included in the official programme: Committee of the First International Congress of Sanatoria and Private Nursing Homes, Budapest, Margitsziget Sanatorium.

GALVANIC IONIZATION THERAPY IN ALLERGIC RHINITIS AS EMPLOYED AT THE MANHATTAN EYE, EAR AND THROAT HOSPITAL.*

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The purpose of this paper is to relate our experiences at the Manhattan Eye, Ear and Throat Hospital in the treatment of 45 cases of allergic rhinitis, since June, 1935, with galvanic ionization. The treatments have been conducted in the clinic of Dr. Francis White under the supervision of Dr. Louis Hubert. Some earlier work with this mode of therapy at this hospital indicated that the method had merit and a number of articles on the subject by various investigators in other places appearing in medical publications and expressing varying degrees of enthusiasm seemed to justify further investigation on our part.

A good deal has been written on the subject of apparatus, but we believe that nothing of an elaborate nature is required for this work. The first outfit which we used cost several hundred dollars, but at present we are using a machine of an inexpensive dry cell type employing two 22½ volt radio batteries. A cell selector permits the use of 7½ volts and multiples to the capacity of the machine and an efficient rheostat gives nice regulation of the current without any jumps. It is fitted with a milliampere-meter and a volt-meter. The former indicates the amount of current being used and the latter the potential or pressure. It serves also to indicate the condition of the batteries. We started with the total of 45 volts and today, after approximately 100 treatments have been given, it registered 35 volts. It is probably good for many treatments. The batteries cost $1.80 and the whole machine, including the batteries, $22.50.

A variety of intranasal electrodes have been employed by various experimenters. We have tried out cotton, cellulose, and jelly. In the earlier work, the Warwick metal electrode

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was used in all cases, but in this series now being discussed, it has been employed only a few times; for it has been found that the cotton, saturated with an electrolyte, serves quite satisfactorily as an electrode when carefully inserted. We have used Warwick's zinc, tin, and cadmium solution, but for some time now have been using only the zinc sulphate solution as described by Friel, Cottle and others. It is used in strength from ¼ per cent to ½ per cent. Preliminary cocainizing of the mucous membrane has been found advisable. The cotton is carefully inserted into the nasal cavity so that all parts of the mucous membrane are covered by it, with the exception of the olfactory nerve ending area. About one-half inch of cotton electrode is allowed to project outside of the nostril and to this the clip of the cord leading to the positive pole of the battery is attached. It has been found that if vaseline is smeared on generously at the entrance of the nostril and on the upper lip, there is no danger of producing burns. An additional safeguard against burns is the placing of a small piece of adhesive plaster over the upper lip so that the electrode cannot come in contact with it. The wire lead is fixed to the forehead with a piece of plaster. Very light rubber-insulated leads are used so as to avoid the necessity of a head band. The indifferent electrode is a linen-covered asbestos fibre pad with a rubber backing to protect the clothing. This is placed at the back of the neck after it has been moistened with a weak sodium bicarbonate solution. This is found somewhat less irritating than sodium chloride solution.

Some of the writers on this subject have discussed the matter of the patient's electrical resistance and some of the machines are provided with an ohm meter. Ohm's law teaches that current varies directly as the voltage and indirectly as the resistance, and the formula is E equals V divided by R, or R equals V divided by E. Now if we know any two of these factors we can derive the third. Let us say, for example, that the voltage employed is 25 volts and the milliamperage 10; the resistance can be easily determined by dividing 25 by 10 and multiplying by 1000, which in this case is 2500 (ohms). It has been found in our work that a resistance around 3000 ohms indicates a normal operating condition.

It was decided at first to use very small doses so as to avoid, as far as possible, producing any injury to the mucous

membrane. A certain amount of untoward reaction is unavoidable. We had in mind the avoidance of an atrophic condition of the mucous membrane which might be produced if repeated large doses were employed. We, therefore, used at first a dose of 5 ma. for a period of 10 minutes, that is, a total of 50 ma. minutes; one nostril was treated at a time, the interval between treatments being about one week.

The reaction following treatment varied considerably in different individuals and even in the same individual. There is no way of predicting the degree and character of reaction. On removing the cotton electrode one may frequently observe a deposit, a sort of grayish membrane. In most cases this is entirely absent, but just why it should be present in one case and absent in the next is not easily explained. However, the presence or absence of such a membrane seems to have little bearing on the ultimate effects. There is frequently some immediate relief from the clogged condition in the nostrils and breathing is made easier for the time being at least. Within a short time, however, a matter of an hour or so perhaps, the clogged condition usually reappears and persists for several days. The amount of discomfort suffered by patients varies considerably. Some have practically none, others have a great deal of pain or aching of a neuralgic character in the side of the face treated. It may also extend into the opposite side.

Attempts have been made by various writers to explain the modus operandi of galvanic ionization, but inasmuch as our knowledge of the action of electric currents on tissues is very incomplete, it seems unwise to engage in more or less idle speculation at this time. It is a very complex electro-physical-chemical study, and from the practical therapeutic point of view the method of trial and error, by which most, if not all medical and surgical procedures are established, is unavoidable.

We are convinced that this method of treating allergic rhinitis is a real boon to a percentage of sufferers from this condition; but, frankly, we do not know just how valuable it is, as present available evidence is very conflicting. This series, we believe, is not presented with any unwarranted conclusions. It is a small series and covers only a short time, and our technique is in an evolutionary stage. Many of the cases have had various forms of treatment before coming to us, such as operation,

desensitization injections, chemical cauterizations, etc. Some have had an associated sinusitis and we are of the opinion that good results are not to be expected in the presence of this condition. Ionization, as we have used it intranasally, has not helped the infected sinuses in any way.

Coming now to actual results, they may be divided for simplicity into immediate effects, that is, within a period of two or three weeks following the treatments, and the later results. As stated before, the degree of reaction varied considerably on the whole, but only in a small percentage could it be called severe. During the second and third week most of the patients felt distinctly better. There was less sneezing, the amount of secretion was reduced, breathing through the nose was easier, and life was much more tolerable. In a few cases, however, there was very little, if any, change for the better. This is especially true of patients suffering from an associated sinusitis. About 10 of the cases have been treated so recently that we cannot form any opinion about the result of ionization as yet. Of the remaining 35, most of them have been seen within recent weeks, and in a fair number of cases considerable benefit has been derived. Two of them are symptom free; one, the first case that we treated, having suffered for 12 years. In a few of the cases no benefit, either immediate or late, was derived.

Summarizing, it may be said that this form of treatment is good in some cases, of moderate value in others, and without benefit in the rest, but we feel that it will take considerably more investigation over several years before its true worth will be known.

Manhattan Eye, Ear and Throat Hospital.

SURGICAL APPROACH TO THE NASAL ACCESSORY SINUSES.*

DR. WM. MITHOEFER, Cincinnati.

There seems to be a diversity of opinion among rhinologists regarding the proper surgical approach to the nasal accessory sinuses. This is probably one of the reasons why nasal sinus surgery has fallen into disrepute. It is indeed a sad commentary on modern rhinology to see patients who have been subjected to four or five nose and sinus operations, without getting relief, and to find later that the chief offender has been a chronically diseased antrum which should have been radically cared for at the time of the first operation. Too many nasal operations for the relief of obstruction are being done without giving the nasal sinuses the consideration they deserve, prior to the operation on the nose. It is our contention that a submucous resection of the septum, for instance, should not be done without first making a careful study of the nasal sinuses, including X-ray examination. It is certain that a middle turbinectomy should not be performed in the presence of a chronic ethmoiditis; rather preserve the middle turbinate and exenterate the "key to the situation" — namely, the ethmoid cells.

It is well worth remembering that a general dyscrasia has very often a disease of the nasal sinuses as its accompaniment, the most frequent diseases being hypothyroidism, chronic intestinal toxemia, gouty-arthritic diathesis and diabetes. Many a patient with a mild nasal sinus disease is made more comfortable by directing attention to the imperfect drainage system in the colon, and at the same time supporting the thyroid gland with a tolerant dose of thyroid extract, because this gland has usually ceased functioning adequately in consequence of the chronic toxemia originating in the colonic cesspool.

Hypothyroidism of the benign type with a subnormal temperature between 4 and 6 P. M., slow pulse, low blood

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pressure and minus basal metabolic rate is a frequent disease in adults, as well as in children. As a result of insufficient action of the thyroid gland, infiltration of mucous membranes takes place, the nature of the soil changes, and bacterial invasion is more apt to occur. Patients of this type react poorly following an operative procedure of any kind. The tolerant dose of thyroid extract is reached by beginning with small doses, and increasing slowly until the afternoon temperature reaches normal or the pulse rate exceeds 95 beats per minute. When once the tolerant dose has been ascertained, this dose should be given three weeks of every month, with an occasional rest period of longer duration. A biochemical examination of the blood may be of diagnostic importance in some cases, inasmuch as an increase of the uric acid or a decrease of the sugar content of the blood are dyscrasias which add fuel to the fire in patients suffering with nasal sinus disease.

Examination of the nose and the adjoining air cells should be a very comprehensive one, before proceeding with the operation. All diagnostic methods at our command should be used, especially in those patients in whom there is a mild involvement of the sinuses which may not require operative removal to obtain relief. It is otherwise, however, in patients with extreme polyposis or intraocular or intracranial complications. In the diagnostic study of our patients, we have repeatedly observed relief of headache after infraction of the middle turbinate (median rhinoscopy), followed by cauterization of the lateral wall of the nose opposite the middle turbinate with trichloracetic acid. There may or may not have been present a mild ethmoiditis in these cases.

If we are dealing with an allergic patient, we do not hesitate, if diet and other conservative means have failed, to proceed with the operation on the nasal sinuses, at the same time removing any intranasal obstruction which is preventing free nasal respiration. These patients are, of course, not always relieved of their constitutional allergic manifestations, but we have found that after operation the nasal symptoms are present to a much less degree. We do not advocate this procedure except in well studied and extreme cases.

The two questions to be decided by the rhinologist are: *1.* When shall we operate? *2.* How shall we proceed with the operation?

To know when to operate is a very difficult question to decide. There are so many patients who have symptoms referable to the nasal sinuses, but in whom there is found little, if any, pathological change in the nose. For example, a patient complains of a profuse postnasal discharge, especially in the morning hours. Nasal examination reveals nothing of any consequence. A more careful analysis, however, gives us the following information: Skiagram of the antrum reveals a slight haziness with indistinct margins. Lavage of the antrum is negative. Lipiodol test shows a partial filling defect. It is also ascertained, while probing the lateral wall of the nose, that there is present a large accessory ostium which permits free drainage of mucus upon arising in the morning. A diagnosis of chronic serous catarrh of the antrum is made. A radical antrum operation is done and the antrum cavity, as well as the deep recesses, are found to contain edematous tissue. The ethmoid labyrinth is opened through the transantral route, and a serous inflammatory process is found in all of the cells. Failure in diagnosis may readily occur if all factors are not given due consideration in a patient of this type. The question may arise in your minds whether it was necessary to do a radical operation, since a window resection might have answered just as well. It has been our experience that a chronic serous inflammation of the nasal sinuses can only be removed in an operative way, and that all vestige of diseased mucous membrane must be eradicated. Unless this is done, the discharge continues as before. We are fully convinced that such is the case, inasmuch as we have seen many patients whose sinuses were chronically involved with a serous inflammation, and upon whom a window resection had been done elsewhere without benefit. In the suppurative variety of antrum inflammation, the opposite may be true. These patients may be relieved following an intranasal antrum operation. So that in making our surgical approach to the nasal sinuses, we must also consider the type of inflammation which is present, if we are to proceed in a proper surgical manner.

The most difficult cases to fathom are those in which the examination of the nose is entirely negative. The patient's chief complaint is headache, located in the supraorbital region. A tentative diagnosis of "vacuum headache" is made. There is no constitutional dyscrasia. In a case of this type, we may be

dealing with an anatomical variation of the nasal sinuses with closure of the nasofrontal duct. Dilatation of the nasofrontal duct with Ritter's sounds is unsuccessful. The sound does not enter the frontal sinus, as shown by the position which it occupies, as well as by the skiagram. The latter also shows an upward displaced infundibular cell extending into the floor of the frontal sinus. This is but one of the numerous anatomical variations of the nasal sinuses. There is no region of the body where anatomical variations are of such frequent occurrence, and it is probably this fact alone, in many instances, which is the cause of failure following operation. We have seen a number of patients with an upward displaced infundibular cell where relief of headache was not obtained until the displaced cell was removed by doing an external ethmoid operation. In most of these patients, little, if any, pathological change was found in the sinuses, the headache being caused by closure of the nasofrontal duct. The ethmoid cells may also extend laterally as well as downward over the roof of the antrum. The sphenoid sinus may extend laterally and be separated from the antrum by a very thin plate of bone. It is because of these anatomical variations that a proper decision on how to proceed will always be an important consideration of the nasal surgeon.

The surgical approach to the various sinuses will remain a moot question until rhinologists become agreed on the proper method of procedure. A safe rule to follow is to operate only after a careful consideration and analysis has been given all factors, both local and general.

The first thing we always attempt to decide is which cavity is the chief offender, and whether the disease has involved other air cells as well. Many times it is necessary to begin operating in the antrum, and through the transantral route reach the ethmoid cells, preserving the middle turbinate, if possible. This method of attack will very often give excellent results. We are not in favor of operating upon the ethmoid cells extranasally as a first requisite, the exception, of course, being a marked pansinusitis, especially if the patient happens to be an asthmatic. We try to decide before operating which are the affected cavities, and then deal with all cavities at one sitting if it is at all possible. We have found that the patient has less reaction with such a procedure; certainly much less than when an incomplete operation has been done.

One of the causes of failure following a radical antrum operation, for instance, has been the fact that disease of the ethmoid sinuses has been allowed to remain, the supposition being that disease in this region will care for itself after the antrum disease is eradicated. Such an outcome, however, is of rare occurrence. The antrum continues to be a reservoir for the discharge from above. It has been our experience to find an involvement of at least the anterior ethmoid cells in most cases when operating upon a chronically diseased antrum. The disease in the ethmoid may be of a different pathological character than that found in the antrum, the ethmoid being involved with a serous type of inflammation, whereas the purulent or mixed variety is found in the antrum. The same is true of the frontal sinus in the presence of a marked, chronic, purulent inflammation of the ethmoid cells. We have found many times while doing an external ethmoid operation for the relief of suppuration, that the frontal sinus did not contain pus, but instead was filled with a serous exudate with the mucous membrane soft and edematous. This has a correlation with hydrops of the knee joint in the presence of inflammatory processes of the epiphysis.

We may rightly say that the ethmoid sinus is the "key to the situation" and requires investigation whenever a sinus operation is performed. Many times the ethmoid cells alone are involved, and an ethmoid operation, with the preservation of the middle turbinate, will often be of good consequence. It is impossible, however, in most instances to remove all of the ethmoid cells intranasally, especially if there are upward and lateral prolongations. Downward prolongations, the so-called inferior hiatus cells, can often be reached by removing a part of the middle meatal wall of the antrum, in this way also giving better ventilation to a mildly involved antrum cavity. We are of the opinion that an opening to the antrum in the middle meatal region is better placed for aëration of the cavity, because this region is in line with the inspired air current. An enlarged opening in this locality will also allow for better exit of secretion, inasmuch as it has been shown that the ciliary action of the antrum mucosa is towards the normal ostium. It is true that an opening in the antral wall, above the inferior turbinate, is not as well placed for drainage purposes as is one placed under the turbinate; but neither does an infraturbinal window allow for sufficient drainage when the maxillary sinus contains deep recesses.

Rhinologists who advocate intranasal antrum operations, and prefer this operation to the more complete procedure, such as the Caldwell-Luc and ethmoid operation, report relief of symptoms in a great number of patients. If headache was the chief complaint, relief is easily obtained with an intranasal operation, because of the removal of pressure and the procuring of better drainage. It is doubtful in our minds if these patients are freed of their chronic toxemia, when large areas of diseased mucous membrane are allowed to remain. Acute flare-ups are frequent in these cases following a nasopharyngitis or a general systemic derangement, especially one originating in the colon. There is less danger of allowing a hidden sepsis to continue its systemic invasions if a complete operation is performed. We do not wish to imply that intranasal antrum operations have no place in surgery. Our claim is that they should not be done as a routine measure on all diseased antrum cavities. After careful analysis has shown that marked pathologic changes are not present in the antrum, or if there is a pure anaërobic or suppurative infection, we may well proceed with this type of operation. A window in the antral wall will certainly have no influence on a chronic serous inflammation. At least, this has been our experience.

It happens occasionally that after a well done, complete nasal sinus operation, the patient continues having pain in the region of the antrum and behind the eyes, with radiation of pain in various directions. We are apt to think of an affection of the Vidian nerve or sphenopalatine ganglion in these cases. If instillation of cocain into the sphenoid cavity, or if the application of cocain to the sphenopalatine area does not bring relief, we must exclude the presence of a "silent osteitis" of the alveolar process, or an osteitis of the frontal or sphenoid bone. A latent osteitis in these areas is easily overlooked. If the alveolar region is carefully palpated, it may be found painful on pressure and slightly thickened. Pressure of the examining finger may excite a severe attack of pain. If such is the case, we are undoubtedly dealing with a case of latent osteitis of the alveolus which should be approached by completely removing the diseased bone. This requires the services of a dental surgeon.

It behooves us to remember, therefore, that a latent or, as it is sometimes called, a "silent osteitis" of the alveolus may be

the cause of continuance of pain after a well done radical antrum operation.

In conclusion, I wish to consider briefly the various methods of surgical approach which we are in the habit of using.

1. Dental antritis — extraction of tooth followed by irrigation of antrum by the rhinologist, and not the dentist. Allow the alveolar opening to close. If the irrigating fluid is still positive after eight or ten lavages, lipiodol should be instilled. If there is present a filling defect and the secretion is still fetid, a radical antrum operation is given due consideration. If no filling defect is present, intranasal antrum operation may be the method of choice.

2. Antrum cavity acting as a reservoir. Lipiodol shows no filling defect. In these cases we remove the ethmoid cells intranasally, and also the greater portion of the middle meatal antral wall with preservation of the middle turbinate if possible. If a filling defect is present, the antrum is explored before doing the intranasal ethmoid operation.

3. Polyposis with cloudy antrum and ethmoid. Radical antrum, intranasal ethmoid operation; if necessary external ethmoid, frontal and sphenoid. External frontal operation is many times done only as a last resort. Lipiodol test is usually unnecessary, inasmuch as we generally find an antrum involved in cases of polyposis with cloudy skiagram.

4. Dentigerous and other cysts of the antrum — radical antrum operation.

5. Negative nasal findings; skiagram negative; lipiodol test shows no filling defect. Patient complains of fulness in the cheek — exploration of the antrum through the facial wall. In these cases we have many times found pathological changes in the recesses, with edematous swelling of the mucous membrane in the region of the normal ostium.

6. Antrum suppuration; no filling defect. Many times anaërobic — intranasal antrum operation for ventilation.

7. Serous inflammation of the ethmoid—intranasal ethmoid operation. External operation may follow if patient is not relieved, and if the X-ray shows lateral prolongations of

ethmoid cells into the roof of the orbit. Exploration of the antrum if there is any doubt as to the presence of pathological changes in this cavity.

8. Suppurative ethmoiditis — intranasal operation. If pus exudes from the nasofrontal duct at the time of operation, follow immediately with the external frontal. There is less danger of osteomyelitis as a complication if this is done.

9. No pathological changes found in the nose; bulla ethmoidalis in apposition with large uncinate cell causing closure of nasofrontal duct (vacuum headache) — intranasal frontal operation.

10. At the time of ethmoid operation, region of the sphenoid should be investigated. If this cavity is found diseased, or if the normal ostium is closed by thickening of the anterior wall, sphenoid operation follows. If the lining membrane of the sphenoid cavity is found involved, same is carefully removed.

11. Upward displaced infundibular cell causing closure of the nasofrontal duct. If unable to open nasofrontal duct satisfactorily in doing the intranasal operation, external ethmoid may be indicated.

12. Radical external frontal operation when intranasal operative measures have failed to relieve the patient of headache.

13. Radical frontal sinus operation in presence of orbital, intracranial complications, or osteomyelitis.

14. In the fulminant type of infection, use conservative means for a few days and, if no relief is obtained, proceed with radical ethmofrontal operation.

15. If "silent osteitis" is suspected in the alveolar region, consultation with dental surgeon. If the sphenoid bone is involved with osteitis, removal of the entire anterior wall of sphenoid. A latent osteitis of the frontal bone should not be operated upon unless symptoms demand.

Again, let me emphasize the importance of thorough analysis of each patient before taking the operative step, and at the time of the operation to determine which cell is the chief offender and whether the discharge is originating from an

involvement of a combination of cells. It cannot be too strongly emphasized that all sinuses found diseased should, if possible, be surgically approached at the first operation.

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AMERICAN LARYNGOLOGICAL ASSOCIATION.

The fifty-eighth annual meeting of the American Laryngological Association will be held at the Book-Cadillac Hotel, Detroit, Mich., Monday, Tuesday and Wednesday, May 25, 26 and 27. The first session will begin at 10 A. M. on Monday, May 25.

The Council recommended for election to Emeritus Fellowship, Dr. Joseph L. Goodale, of Boston; for election to Honorary Fellowship, Dr. Herbert Tilley, of London, England; for election to Corresponding Fellowship, Dr. John G. Fraser, of Edinburgh, Scotland; and for reinstatement to Active Fellowship, Dr. John L. Barnhill, of Florida.

The American Bronchoscopic Society meeting will be held on May 27, and will be followed by the meeting of the American Otological Society on May 28 and 29. All of these meetings will be held at the Book-Cadillac Hotel.

THE OPHTHALMOLOGICAL RELATIONSHIP TO THE NASAL ACCESSORY SINUSES.*†

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From an ophthalmological standpoint, there are three points to be discussed: The first is relative to the etiology or manner of development of the ophthalmological complications; the second is to mention the various complications which occur; and the third, to consider in some detail the more important of these complications.

As to the first of the above subdivisions, various possible anatomical possibilities must be most carefully judged; and of equal importance is the role which toxins (or perhaps allergens) must also play. Experiments have shown that micro-organisms placed upon the nasal mucosa penetrate into the vitreous and aqueous of the same side without causing signs of general infection, hence the conclusion is warranted that bacteria penetrate directly into the eye from the nasal and the conjunctival mucous membranes. Experiments with India ink show the exact route followed by such particulate matter from the conjunctival lymphatics, along Schlemm's canal and into the anterior chamber, last into the vitreous; Kistner,[1] working with nasal sinus infections, as associated with diseases of the vascular system, found that in a series of 400 sinuses, mostly chronic nonsuppurative cases, the fresh tissue taken at operation and ground, washed and cultured under aseptic precautions showed streptococci to be present in over 90 per cent of instances. Staining for organisms in the tissues, a diplococcus, with the staining characteristics of the streptococcus, was found most commonly lying in the deeper tissues or layers of the membranes. With the idea that there might be a selective action of the organisms for blood vessels, the sinus tissue was sectioned and stained in 18 cases. In 11 of these, organisms were found in the thick-

*Read before a Joint Meeting of the Ophthalmological Section and the Oto-Rhino-Laryngological Section, the College of Physicians, Jan. 17, 1936, Philadelphia. This paper was the ophthalmological viewpoint in a Symposium on the Nasal Accessory Sinuses.

†From the Peter Clinic, Graduate Hospital, the University of Pennsylvania, Graduate School of Medicine, Philadelphia.

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ened vessel walls and accompanied by cell infiltration characteristic of chronic inflammation. The importance of this invasion of the vessel walls is readily seen when considering that at least one-third of the venous drainage of the entire nasal mucosa passes upward into the ethmoidal veins. These have an active communication with the ophthalmic veins and with the veins of the dura. The superior ophthalmic vein has a rather direct connection, while the inferior ophthalmic vein has a less direct but much more extensive connection with all the other nasal veins through its terminations with them in the pterygoid plexus. In addition, the entire plexus of veins about the lacrimal sac anastomose with the ethmoidal veins. A recent case of multiple, in fact innumerable, metastatic sarcomata in the choroid and the retina from an original neoplasm on the forehead seems to conclude that vascular paths for the conduction of infected material exist and do duty as such. It seems that the venous circulation is the greatest factor in the mechanical transportation of pathological products.

As to toxins, it is rather likely that this is of no less importance. It is not necessary, nor likely, for both to be present in the same case, but certainly, in a case of bilateral frank papillitis, not retrobulbar but bulbar, connected with ethmoidal empyema in both the anterior and the posterior cells, the role of one is as possible and probable as the other. Rosenow and Nickel,[2] while working together on elective localization in chronic uveitis, showed this. Elective localization in laboratory animals of a streptococcus (alive) or the toxins (dead organisms and filtrates of cultures) has been obtained in a sufficiently high percentage of cases, by methods sufficiently variable, and with conditions sufficiently alike those at hand in patients, to be of indubitable significance. The importance of the pure toxin has been well demonstrated by Brown.[3] In his work on the experimental production of uveitis he was working wholly in anaphylaxis and without live organisms. He produced an acute inflammation of the uvea by the intraocular injection of a streptococcus toxin, which inflammation subsided and recurred after the intravenous injection of the same toxin 10 days later. He was endeavoring to produce recurrent attacks of uveitis which would be toxic in character and nontraumatic in effect. He concluded that the ability of a toxin to sensitize local ocular tissues is

significant, in that it may be possible for a focus of organisms to eliminate some absorbable substance into the circulation and so sensitize the uveal tract that the recurrences of uveitis might represent periods of hypersensitiveness and the remissions periods of desensitization. I would like to quote Jackson[4] relative to these etiologic factors: "In a single generation the etiology and prevention of disease has advanced to the leading interests in the science and art of medicine. As to the etiology of uveitis, the literature, of the last 30 years outweighs in value that produced in all preceding generations."

The limits to which the ocular complications of sinus pathology can extend are still almost uncharted. Two cases will serve to illustrate these extremes. The first was that of a man, age 35 years, with dizziness, bilateral conjunctival injection, photophobia, and with pain with all movements of the eyeballs. This group of symptoms had been present for slightly less than 24 hours. The diagnosis, that of an acute Tenon's capsule inflammation, was probably correct. He had had an acute rhinitis 48 hours before with apparently an early extension of the infection into the maxillary antra bilaterally; in addition, the inferior turbinates were reported as being markedly swollen and congested. After appropriate treatment by a rhinologist, he was ocularly symptom-free in another 24 hours. His antral infection was cleared up within 10 days, but from the time drainage was first established he had had no other eye signs or symptoms. The second case was one of frontal sinusitis, bilateral, following after a two to three weeks' illness with influenza. The patient had been out three rainy nights in succession, before full recovery and while still running an evening temperature. He again became quite acutely and severely ill. Transillumination, when first seen by a rhinologist, showed bilateral dark frontals with cloudy anterior ethmoidals. Within the next three days the patient developed serious signs of a diffuse meningoencephalitis. Upon examination he showed a bilateral acute iritis, a bilateral frank papillitis, more marked in the left eye, paralysis of both external recti, bilateral lagophthalmos from double facial paralysis, limitation of upward rotation in both eyes and a paralysis of the levator upon the right side. The lagophthalmos, upon the left, which was not present to the same degree upon the right because of the ptosis there (and fur-

ther, made worse on the left by the levator paralysis), made tarsorrhaphy necessary to check a rapidly developing exposure keratitis. In addition to these eye changes, the patient had a bilateral involvement of the Vth nerves (his teeth felt as if laid in cement), bilateral involvement of the vestibular branch of the VIIIth nerve, paralysis of the postpharyngeal muscle, involvement of the XIth cranial nerve and of the brachial plexus (weakness and numbness of the upper arm), cardiac symptoms and a profound constipation akin to paralytic ileus (stomach X-rays showed a high degree of retention and almost completely absent peristalsis), *i.e.*, Xth nerve, and both taste and smell were to some immeasurable extent definitely blunted. While there is no doubt that the encephalitic process was responsible for the extent of these cranial nerve symptoms, Stout,[5] who presented the case, felt that the various individual infectious ocular conditions, as well as the meningoencephalitis, were not purely influenzal, but that the pansinusitis was equally to blame. Ophthalmologically, this opinion can be confirmed, in that the patient developed both possible types of complications. The iritis was undoubtedly a local infection from the focus of the sinuses, the oculomotor paralyses and the papillitis could have been either central or local (focal) in origin, while the lagophthalmos was due to a central type of VIIth nerve paralysis. The accompanying left keratitis was, of course, mechanical in character, though a possible Vth nerve hypesthesia might have been an additional factor. The patient made a complete recovery. A bacteriological study of this case would have been most interesting, if this could have been done. War-time experiences seemed to show that a secondary infection, when present, in addition to the influenza bacillus, aggravated influenza to a very serious degree, and especially the resulting complications; even to being the immediate cause of such complications. The pneumococcus, as well as the streptococcus, have appeared in such instances about equally often. Rather likely, this was the sequence of events here.

The various complications which have appeared can be readily grouped anatomically. The uveal tract, including iritis and iridocyclitis, retinitis and chorioretinitis; the muscles of the ciliary body, certainly insofar as cyclitis is concerned, though accommodation paralysis alone seems to be rare; conjunctivitis, episcleritis, scleritis and inflammation of

Tenon's capsule; the cornea; orbital and retrobulbar conditions, the extraocular muscles; and the optic nerve, both in its bulbar as well as its retrobulbar portions. Conditions distantly related, but still indirectly connected, would include the ocular manifestations of such conditions as cranial osteomyelitis, intracranial infections, extradural and subdural abscesses, meningitis and encephalitis, and cavernous sinus thrombosis. These all may be dismissed with this bare mention, though not from a consideration of their seriousness. Too often, both the ophthalmologist and the rhinologist think of nothing but the optic nerve instead of these various possibilities. They must not lose sight of the other serious and important complications in their search for optic nerve changes.

Inflammatory conditions of the uveal tract due to sinus pathology are not rare. Such an etiological opinion may be expressed only after careful investigations and the elimination of all other possible causes. With the various writers, the incidence of it seems to be equal within reasonable limits. Gifford,[6] in 1931, gave some of these rates as follows: Gilber, 4 per cent; Bulson, the same; Brown and Irons, each 2 per cent; Newton, 8 per cent. Gifford's own series of 118 cases gave a percentage of 6.8. This was one of the largest series and is therefore significant. Unilateral uveal disease is apparently more common than is bilateral, though in the Peter Clinic a fair percentage of bilateral cases has been seen. Several of these had a higher degree of pathology in one eye than in the other. In many of these cases, it seems as if one other factor is rather important in the precipitation of the disease, in addition to the sinus pathology. This is in the nature of trauma or of some mild irritation, local or general, not of itself (under ordinary circumstances) sufficient to cause the uveitic process. To mention a few is adequate. Severe unilateral iridocyclitis in a young man, age 26 years, with ethmoidal and sphenoidal pathology, following several days after an embedded foreign body was removed from the cornea. Severe iridocyclitis in a man, age 55 years, following a successful retinal detachment operation, who developed an influenzal infection while still in the hospital, pansinusitis, pneumonia and, to complete the tragedy, uremia following diabetic coma. The case recovered, but with a hopelessly lost eye. Bilateral iritis with severe descemetitis in a man, age 45

years, with ethmoidal and sphenoidal pathology, following several very hard and tiresome days of travel and preparation for a scientific exhibition. Exudative chorioretinitis in a woman, age 45 years, with chronic sphenoiditis, in whom alcohol, tobacco and dietary indiscretions were very much in the foreground. Bilateral severe hemorrhagic punctate or purpura-like chorioretinitis in a woman, age 52 years, with bilateral frontal sinusitis and with essential arterial hypertension (if such a pathological entity exists). Further, there exists a relationship in uveitis to tuberculosis which cannot be lightly dismissed. In 1917, Luedde,[7] in discussing a paper of Peter's[7] on the use of tuberculin, together with him, cited a series of eye conditions of tuberculous origin, which failed to respond to tuberculin treatment until the posterior group of cells had been thoroughly opened and drained. After this process, as a rule, response to tuberculin was prompt. Naturally, these various factors may be of importance, not through irritation *per se*, but only through a further lowering of an individual's resistance. This fact, however, is outstanding. If we agree that the incidence of sinus pathology is far in excess of the frequency of the ocular complications which appear, this is especially so in considering the uveitic. retinitic and chorioditic complications.

That the ciliary body shares in the inflammatory process of the anterior uveal tract is quite evident. It is not at all unlikely that internal ophthalmoplegia has been seen also. The writer has seen two cases of possible accommodation paralysis, which were in both instances accompanied by wide dilation of the irides. The first was a 10-year-old girl with postinfluenzal bronchitis and pansinusitis. She complained of a sudden loss of near vision while reading during her convalescence. There had been several short attacks, well described by the child and by the mother, and during the last of these she was seen personally. The second was a physician, moderately myopic, who during an attack of ethmoiditis complained of periods in which his pupils dilated, both distance as well as the near vision became blurred, and for an hour or so after he felt dizzy and lightheaded. It is rather interesting that both of these cases had some tenderness upon pressure over the inner upper angles of the orbita, the so-called Ewing's sign, similar to so many other signs in medicine, frequently discussed but not as commonly found.

Pathology in the conjunctiva is quite common but it is seldom if ever purulent. It seems to be a part of a condition which for want of a better word may be called asthenopia. Scleritis and episcleritis have been described, but so also has glaucoma and cataract, as being due to sinus pathology. Rather recently the writer saw a case of chronic scleritis, bilateral, with a low grade chronic ethmoiditis and sphenoiditis; but the patient was, in addition, mildly diabetic and had both clinical as well as Roentgenological signs of old pulmonary apical pathology. Tenonitis seems to be frequent and to have a similar relationship to sinus pathology as does conjunctivitis or conjunctival irritation. Pain and tenderness when pressure is applied to the eyeballs and with movement of the eyeballs is a common complaint. Pathology of the cornea is not a clear-cut complication. Superficial punctate keratitis has been considered as a result of focal infection. Naturally, the deep keratitis of iridocyclitis and descemetitis are to be included in inflammatory conditions of the uvea, but not as complications of sinusitis, only as additional involvements of the uveitis.

As to the various orbital complications, unfortunately we all are rather well acquainted with retrobulbar inflammations, with cellulitis, with retrobulbar phlegmon, and with retrobulbar abscesses. Relative to the last condition, one of the most interesting phases of it seems to be the inevitable controversy between the ophthalmologist and the rhinologist: shall drainage be established through the eyelid externally, or through the ethmoids internally? Use both routes, if necessary, but the drainage must be instituted. Cavernous sinus thrombosis, orbital periostitis, orbital osteomyelitis and meningitis are additional complications, so grave in consequence that every possible effort should be put forth to prevent them. As it is, even with the best of attention and effort they occur at times. Dowman's[8] and Sir St. Clair Thomson's[9] cases of mucocele of the frontal sinus, Ferreri's[10] pulsating mucopneumatocele of the frontal sinus, Reverschon's and Worms'[11] and Bower's[12] cases of mucocele of the ethmoid, Hajek's[13] case of mucocele of both these sinuses, and Peter's[14] case of exostosis or periostitis of the ethmoid are all inclusive and classical examples of these somewhat rare complications. Acute suppurative dacryocystitis, it is almost certain, has been caused by an extension of ethmoidal pathology, but this can-

not be conclusively proven. The course of events in some of these cases is more than suggestive, however.

The extraocular muscles are repeatedly involved, and in some of these cases the improvement and cure which follows shortly after sinus surgery is almost miraculous. One or both internal recti, the external recti rather commonly (according to some, most commonly), the superior oblique, the superior rectus and the levator, the inferior oblique, in fact all of the ocular muscles, have shown frank oculomotor paralyses or less complete pareses. Further, with the phorias, there is but little doubt that exophoria at least, if not caused by sinus pathology, is certainly in some cases most definitely aggravated by it. Three cases, briefly described, will illustrate sufficiently. The first, a young lady of about 24 years, had a frank left-sided paralytic divergent strabismus. Twenty-four hours after an ethmoidectomy and sphenoidectomy, the diplopia had started to recede, and within three days the patient was again without diplopia. The second was a left inferior oblique partial paralysis or paresis in a woman, age 35 years, with left maxillary antrum pathology, which receded wholly in a very few days following a radical sinusectomy of the antrum. This case had a similar experience upon the right side several years before. Both of these cases were on L. C. Peter's service at the Graduate Hospital. The third, a neurotic young lady, age 22 years, with exophoria for both near and distance, was quite uncomfortable, in spite of active orthoptic training for the phoria until a chronic generalized sinusitis was cleared up by continued nasal treatments. Only then did her phoria show recession. As to the manner of attack, in these cases, one may conjecture thereon, whether it is pressure upon the nerves, or actual invasion of the nerve by toxins or bacteria, without denite conclusions. Rather likely, as mentioned under uveitis, both factors play a part, though under different circumstances. The temporary trochlear paralysis seen commonly after frontal sinus surgery remains at times permanently, so it cannot be too lightly dismissed. Ohm[15] reported three such instances.

The last of the ocular complications to be considered are those connected with the optic nerve. This is to include both the bulbar as well as the retrobulbar portions of the nerve. As one reads through only a portion of the voluminous literature of this subject he is confused and confounded by the

sincere differences of opinion which exist. This does not apply to the cases of papillitis, or optic nerve neuritis, acute, with clear-cut ophthalmological changes, grave visual defects, both in the central as well as in the peripheral fields, and with definite inflammatory signs in the nasal accessory sinuses, especially those of the posterior group of cells. It applies to the cases of retrobulbar neuritis, with but few ophthalmological changes, and with the major number of signs and symptoms in central visual acuity, and in the central and the peripheral fields of vision. The differences of the European ophthalmologists are no exception. Weill,[16] of Strasbourg, said: "The prognosis of neuritis retrobulbaris acuta, even without surgical intervention, may be considered favorable, and the origin should be sought somewhere else than in the posterior sinuses." Van der Hoeve,[17] on the other hand, states: "If a lesion of the nasal sinus is demonstrated where there is a retrobulbar neuritis it must be treated in every case, even in the presence of syphilis. If the sinuses appear normal the prognosis of the optic nerve lesion must be carefully considered in relation to the difficulties and dangers of operation. It is possible that a patient with multiple sclerosis, diabetes, syphilis, tuberculosis, or rheumatism, may also have an affection of the nasal sinuses, and it would be unwise to treat one possible cause and fail to recognize a nasal sinus disease that may be the actual or contributing cause of the optic nerve lesion." P. Watson-Williams[18] stated: "Cases of optic neuritis benefited by the opening and drainage of the sphenoid and posterior ethmoid sinuses, without finding any pus, has strengthened the writer's opinion that an absolutely negative nasal examination should not exclude the possibility of serious sinus infection." Uhthoff's[19] statistics of 100 cases of multiple sclerosis showed that in 50 per cent there were varying degrees of changes in the optic nerve. White[20] reviewed the end-results of 32 operations on the ethmoid sinuses and sphenoid sinus for disturbances of the optic nerve and found improvement in all of the cases except three, in whom permanent damage had occurred. Benedict[21] feels that the surgery in such cases is simply one of protein shock due to the traumatized tissues and the blood clot retained, similar in all qualities to the shock obtained from any type of foreign protein therapy. It is interesting that some time later White[22] reconsidered his findings and thought then that the tonsils, the teeth and other foci of infection might be even more impor-

tant. Benedict,[21] in discussing 225 cases of retrobulbar neuritis at the Mayo Clinic, found multiple sclerosis in 155 of these cases, while sinus pathology was responsible for only one case. Dunphy,[23] Gifford,[24] Peter,[25] Wilmer,[26] Traquir,[27] Herzog (quoted by Gifford), who felt that at least 50 per cent of cases were connected with the sinuses, Damaria,[28] Sargnon,[29] Bell,[30] Vail,[31] Risley[32] and Stark,[33] also strong believers in sinus relationships, and Syme[34] and many others have written in greater and lesser detail on this subject. That it does occur, none deny. Its incidence, however, is a much mooted question. Several mention a form of acute, almost fulminating, retrobulbar neuritis with sinus pathology coexistent, wherein very rapid atrophy, even to complete blindness, occurs, but with this exception: there is controversy, confusion and sarcasm.

The manner of bacterial or perhaps more likely toxic invasion can be answered upon the basis of its anatomical formation. As Wilmer[26] and several others have pointed out, the optic nerve can be properly considered a prolongation of the white matter of the brain. There are no sheaths or cells of Schwann, but there are true glial cells. Hence its reactions to pathology are exactly similar to those of the central nervous system, and not to those of the ordinary peripheral nerves. Cajal's[35] statement is clear: "The nerve cell or neuron is a structure which is exquisitely sensitive to normal or pathological stimuli. It is susceptible to many toxins and poisons that do not affect, or only slightly alter, the other histological elements." This we know is especially applicable to the fibres of the papillomacular bundle. They will stand pressure and vascular changes for a great time, but to toxins there is considerably less resistance. Benedict[21] said: "It is important to remember that the optic nerve will recover from the effects of injury sufficient to cause temporary blindness, whether from outside sources, such as tumor of the brain or from swelling and infiltration of the septal network of tissue within the nerve trunk, . . . before final degeneration begins; . . . this phenomenon constitutes the basis for all forms of treatment of optic neuritis, and is of primary significance in a consideration of treatment of retrobulbar neuritis from whatever cause." It is to be regretted, but physicians are too often faddists in therapy, as well as in other medical and surgical problems. A warning to all of us, at this point, and

relative to this point is proper. Acute retrobulbar neuritis from sinus pathology seems to be a proven clinical entity, but it is equally certain that it does not appear in epidemic proportions.

Anatomically, there can be no doubt that inflammation may be communicated to the optic nerve within its sheath and its foramen from adjacent structures. Perhaps the size of the bony neural canal plays a factor, also the degree of bony covering to the nerve within the orbit and adjacent to the nasal accessory sinuses. Personally, the writer is wishful of leaning toward van der Hoeve's views in this matter of retrobulbar neuritis. Further, if multiple sclerosis is either locally bacterial in origin or due to a virus, locally active, a relationship may exist from the standpoint of the etiology of multiple sclerosis between undrained foci of infection, as found in the sinuses, and the onset or initial invasion of the central nervous system structures. This condition must still be included within that ever decreasing group of diseased condition for which we do not know the cause.

There is, however, so much which can be justly blamed to these sinuses of ours, with proven guilt, that it is unwise to consider them the scapegoat for every ache and pain, or even the unqualified cause of every attack of iritis or of optic nerve neuritis with which we meet in the practice of ophthalmology.

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REFINEMENTS IN TONSILLECTOMY. RADICAL T. AND A.*

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During the middle of the nineteenth century all forms of surgery were regarded as a butcher's job. The evolution from "butchery" to successful artistry in tonsillectomy and adenoidectomy was due to the superb efforts of such masters as Barnes, Coakley, Fetterolf, Fisher, Matthews, Sluder, Painter, and Fowler, who were the pioneers in this phase of surgery. Particularly since 1910, have the latter perfected a more correct technical T. and A. During the last century tonsillotomy, or partial removal of the tonsil, was exceedingly popular. A portion of the tonsil was deliberately left in the throat as an act of deference to its undiscovered usefulness, the result of which was that the tonsillar remnant grew in size, became troublesome, and the patient almost invariably had to have the operation performed again. Today, the surgeon has found that a complete removal, or tonsillectomy, gives better results, and the human body has demonstrated conclusively that it can get along without tonsils and that their proper removal does no demonstrable harm.

It is no exaggeration to say that it would be difficult to find two laryngologists performing the tonsil and adenoid operation in exactly the same manner. After all, the method is of no importance: Any method, or type of instrument used is successful as long as the surgeon takes due regard to anatomical and pathological conditions of the particular case. It is agreed, however, that haste and spectacular celerity are less indicated here than in any other branch of surgery. On the contrary, slow deliberate manner and attention to details are essential, the surgeon being fully acquainted with the precise nature of the region in which he is working. The one-time conception to remove tonsils and adenoids as quickly as possible no longer

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holds good, because we should be impelled by a keen desire to perform a thorough tonsillectomy and adenoidectomy and not a rapid careless procedure. This end-result should be the principal motive of every otolaryngologist lest the operation be justifiably held in disrepute by many of our laity and physicians, because of the frequent recurrences of lymphoid tissue in the tonsillar fossa or in the nasopharynx.

Our contention is that the operation is not a simple one if it is performed conscientiously and thoroughly. With this thought in mind, we have gradually improved our technique to such an extent as to avoid recurrences in most instances. We have catalogued the operation as the radical tonsillectomy and adenoidectomy because every macroscopic strip of lymphoid tissue in the tonsillar fossae and nasopharynx is exenterated, thus eliminating any possibility of recurrence or any nidus for a future focus of infection. This has been accomplished by a refinement or two, which we believe to be innovational in this phase of surgery.

Whenever the pharyngeal nodules give rise to any symptoms, they should be removed thoroughly and as early as possible. Temporizing is always dangerous; and the practice of waiting for an adenoid to atrophy cannot be condemned too severely, as irreparable damage may be done before this takes place. Although the operation for the removal of the adenoids is a simple procedure, it is more difficult to achieve a "clean" result than in tonsillectomy. Very often, failure is due to a tendency of the surgeon to hurry through this part of the combined operation. Previously, adenoidectomy consisted of the removal of this lymphoid mass by means of an adenotome or curette. Regardless of how thorough one may be with these instruments, the total elimination of the pharyngeal mass is utterly impossible, because careful inspection of the epipharynx will frequently reveal two tremendous chains of lymphoid tissue along the lateral walls and clumps of tissue in Rosenmuller's fossa. The reason for this is that these lateral masses are so frequently implanted on the yielding muscular substructure comprised of the elevators of the soft palate. The latter structures provide no purchase to the inflexible mechanical adenotome and the lateral clefts are too narrow to permit the safe use of a satisfactory curette. The former tissue is similar to that of the posterior wall and is grouped around

the gland ducts in the fibrous mucosa. The same conditions that produce the prominent posterior nodules bring about a diffuse infiltration of the fibrous mucosa of the lateral folds, and the resulting lymphoid tissue is similar in every way. Here, however, the individual nodules are fused so as to form a continuous and prominent band of lymphoid tissue running up either side of the epipharynx just posterior to the posterior pillar. They pass upwards behind the palate and are lost in the left walls of the nasopharynx just below the Eustachian tubes. These lateral masses are frequently the causative factor in the many recurrences of adenoids in youngsters. No adenoidectomy is complete unless the above lymphoid tissue is removed. In order to exenterate the lateral chains, the soft palate is pushed upward and forward by means of the suction tip and the tongue is depressed by the assistant. The surgeon, who by this means visualizes the masses, snares them off with a nasal polyp snare right down to the muscular bed. This snare is employed because of its flexibility, which permits its proper application to the lateral niche of the nasopharynx. Rosenmuller's fossae, which cannot be visualized, harbor the other residual lymphoid tissue which must now be removed. The index finger is placed behind the Eustachian tube eminence for protection with the palmar surface on the tubal mound, thus palpating the lymphoid tissue in Rosenmüller's fossa. A Barnes punch is placed behind the nail of the index finger which protects the tube and acts as a guide, to punch off the small clusters of lymphoid tissue in this area. An inspection and palpation of the nasopharynx will now reveal a recess devoid of the pharyngeal tonsil and its ramifications.

The consensus of opinion at present is that the whole tonsil with all the pathological lymph tissues in the vicinity of it, consisting of lingual extension and the stray follicles on the plicae or elsewhere, should be removed during the operation. Unless the latter is complete, the end-result might defeat its purpose. The correctness of this statement is emphasized by the success obtained after a second operation, when the first has proved a failure. Incomplete tonsillectomy may leave the patient in a worse condition than before the operation, because the part left behind scarifies, and if there is any infection present in the tonsillar crypts, these are promptly sealed up, and drainage, which may have existed before the operation, is now interfered with. Several investigators made bacteriologi-

cal examination of stumps left behind at first operation, and it was shown that on the average, tonsil stumps harbored more pathogenic bacteria per gm. tissue than tonsils which were completely enucleated. With this thought in mind, it is imperative that every vestige of the lower pole, which we will call lingual tonsil, be removed from the tonsillar fossa. The tonsil is divided into an upper and lower lobe by a shelf of muscle fibres which stretches from before backward, somewhat below the middle of the tonsillar bed. Fowler, in his splendid book on tonsil surgery, calls this band the tonsillopharyngeus muscle. These fibres are related to the equator of the tonsil at the junction of the upper and lower lobes, which make an obtuse angle with each other, thus prohibiting complete removal of both by any automatic instrument, as a Sluder, or modification thereof. Neither does the dissection-snare method remove both lobes completely because of the different planes in which they lie. Consequently the removal of the lingual tonsil entails a complete step in itself to accomplish the end-result of thoroughness. Most surgeons grasp this lingual tonsil and snare it off blindly with a large tonsil snare. In many instances, large tabs of lymphoid tissue are left behind because of improper exposure and because of the inflexibility of the snare employed. To obtain an effective removal, the assistant depresses the side of the tongue to cause the entire lingual tonsil to bulge towards the midline and the surgeon grasps only the lingual tonsil and no other tissue. Then, by means of a nasal polyp snare which is flexible for the purpose, the entire lingual is removed by placing the wire loop parallel with the border of the tongue at its base, and slowly drawn home without injuring the tongue muscles. In performing this maneuver the lingual tonsil is not tensed too much to avoid cutting into the muscular tonsillar fossa. This latter refinement obviates much of the sanguination in this area, one of the principal reasons for timidity in doing radical tonsillectomies. In no other manner can the radical T. and A. be performed unless the above refinements are included in every procedure. Kaiser, in his book, "Childrens Tonsils In or Out," describes his examination of 1000 tonsillectomized children for tonsillar remains 10 years after the operation, in order to ascertain to what extent an incomplete operation was responsible for failure in postoperative results; 381 out of 1000 showed signs of tonsillar tissue (no account having been taken of lingual tonsil). The result of his investigations

revealed that children whose tonsils were completely removed fared better than those who had tabs left behind.

Every tyro in tonsil surgery has had the rare privilege of doing a uvulectomy with many misgivings. Our suggestion may arouse much controversy but, nevertheless, we recommend the routine removal of the tip of the uvula because the tremendous postoperative discomfort induced by the pendulous edematous uvula is in this manner avoided, thus providing for a much smoother postoperative course. Furthermore, many future inflammations of the uvula are eliminated to a great extent by reducing the size of this structure which occasionally causes reflex coughing, etc. In none of our many cases have we noted any impediment in speech as a direct result of this uvulectomy.

We have described these few refinements in the hope that the long and shameful list of "tonsil grown again" may be reduced to a minimum because regeneration after a well done tonsillectomy and adenoidectomy is an impossibility, since all the tissue elements from which the lymphoid tissue is developed are removed. It is as reasonable to expect regeneration of an appendix after an appendectomy as to look for any regrowths after a radical T. and A.

887 Ocean Avenue.

The American Academy of Ophthalmology and Otolaryngology desires to announce that a Scientific Exhibit will be one of the features during the New York Convention, Sept. 26 to Oct. 3, 1936.

Applications for space should be sent direct to Dr. S. J. Kopetzky (for Otolaryngology), 51 W. 73 street, New York City; Dr. Ralph I. Lloyd (for Ophthalmology), 14 Eighth avenue, Brooklyn.

CONSIDERATIONS IN THE USE OF BARBITURATES IN OTOLARYNGOLOGY.*†

DR. WILLIAM M. FITZHUGH, JR., Philadelphia.

There is much diversity of opinion among otolaryngologists regarding the proper use of barbital and its derivatives. It is for the purpose of eliminating such confusion that this paper is written. Since their introduction into medicine by Fisher and von Mering 31 years ago (1904), their clinical applications have steadily increased. This growing demand has stimulated manufacturers into producing, without sufficient proof of any clinical superiority, innumerable compounds which are so closely related in their biological action that they are superfluous, if not objectionable.

CHEMISTRY.

Although the chemistry of these drugs is not important to the clinician, yet it is felt, that in order to understand their place in therapeutics, a knowledge of their relationship is of value.

Barbital (diethyl barbituric acid), or Veronal, is a product derived from urea by the interaction with the ester of diethyl malonic acid.

All other barbiturates are derived from this compound.

Phenobarbital (phenyl ethyl barbituric acid), or Luminal, has a phenyl group in place of one of the ethyl radicals in barbital.

Amytal (iso-amyl ethyl barbituric acid) and pentobarbital (ethyl l-methyl butyl barbituric acid), or Nembutal, are both related to barbital in that they have a methyl-butyl group substituted for an ethyl. They are both isomers; *i.e.*, both have the same number of carbon, hydrogen, nitrogen and oxygen atoms. They differ chemically only in that amytal has

*Service of Dr. Otto Hirst, Episcopal Hospital, Philadelphia, Pa.

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a methyl group in the 3 position and pentobarbital, or Nembutal, has the methyl group in the 1 position.

Dial (diallyl barbituric acid) has two allyl groups instead of the two ethyl groups in barbital.

Allonal contains a barbital hypnotic (isopropyl-allyl-barbituric acid) combined with amidopyrine.

Barbituric acid and its derivatives are water soluble if combined with an alkali radical. Therefore, for the sake of brevity only the therapeutic value of their sodium salts will be discussed in this paper.

Evipal (N-methyl cyclohexenyl methyl barbituric acid) is one of the most interesting of the newer barbituric acid compounds, and although the formula is more complicated, its relationship can be noted by comparing it with barbital. The sodium salt is used intravenously.

PHARMACOLOGY.

It is necessary for the otolaryngologist to know the pharmacological action of the barbiturates in order to properly administer them. The degree of drug action is represented by sedation, hypnosis, anesthesia and death, depending on the dosage and type of barbiturate given.

Psychological Effect: When light narcosis is induced, the patient reacts with a feeling of warmth and friendship towards his environment and the world in general. There is a definite shift of the emotional state in the direction of elation. He has no trace of fear, but rather one of relief. As narcosis deepens, it is followed by unconsciousness.

Neurological Changes: The action of the drug on the central nervous system appears to be first on the subcortical center, and then a stimulation followed by a depression of the basal ganglia. In lethal doses there is usually considerable capillary congestion of the meninges and the brain. There is degeneration of the parenchymatous cells of the deeper layer of the cortex, brain stem and cerebellum. All the important nuclei are affected. However, some attribute these changes to postmortem degeneration. When light narcosis is induced, the pupils are dilated, the speech becomes thick and slurred, and the gag reflex remains intact. When a larger dose of barbit-

urate is given, the pupils become slightly smaller and fixed the corneal reflex is finally abolished and there is a tendency for the tongue to fall back and gag the patient. The tendon reflexes are variable.

Effect on Circulatory System: In toxic doses, there is a fall in blood pressure which is due to dilatation of the abdominal vessels and paralysis of the heart. It is more marked in intravenous therapy, possibly because of the alkali essential for its solubility, or the excessive concentration of the drug. The pulse rate is usually increased, probably due to depression of the vagus.

Effect on Respiratory System: In larger doses the respiratory system is depressed. In prolonged narcosis death may result from pulmonary edema or from pneumonia.

Effect on Gastro-intestinal System: It has been observed in surgical work that nausea and vomiting are diminished when barbiturates are used as premedication for any general inhalation anesthesia. In small doses, there is no effect on the liver.

Effect on Genito-urinary System: There is no appreciable effect in therapeutic doses upon the genito-urinary system.

ELIMINATION.

It appears that the shorter acting barbiturates (evipal, sodium pento barbital, sodium amytal) are destroyed in the body probably by the liver, but 50 per cent to 75 per cent sodium barbital and sodium phenobarbital are excreted unchanged in the urine. The drugs partially eliminated unchanged in the urine appear to have a longer duration of action than those which are completely destroyed.

It is for this reason, especially in the case of the longer acting barbiturates, that the maintenance doses should be less than the initial dose, otherwise the drug would accumulate in the system, which would soon be manifested by toxic symptoms.

TOXICOLOGY.

The patient may have an idosyncrasy to barbiturates which commonly occurs from long continued use of moderate doses. The symptoms may be divided into mental and skin

reactions. The mental changes are uncertainty, impairment of memory and moral senses. There are two kinds of skin reactions which may occur: *viz.*, first, urticarial wheals, and second, scarlatiniform rash. It is now the consensus of opinion that barbiturates alone do not cause agranulocytic angina, but this blood dyscrasia may be caused, in some cases (relatively few), by their combination with amidopyrine and its derivatives. In marked dosage, acute poisoning occurs, resulting in coma and death.

The most efficient antidote for barbital poisoning is picrotoxin, especially if there is respiratory failure. The dose of this drug is 1/60 to 1/30 of a grain, which can be administered intravenously (in 5 cc. of saline), intramuscularly or subcutaneously, according to the severity of the existing emergency. Caffeine sodium benzoate given intramuscularly is also of value.

CONTRAINDICATIONS.

The tolerance of these hypnotics varies and the patient should be questioned regarding previous medication before their administration. Patients known to be hypersensitive to these drugs, or those suffering from hypertension, general arterial sclerosis and hypotension should not be given barbiturates. Extreme care should be exercised in elderly patients, as well as those weak and emaciated from chronic illness. In all conditions associated with copious expectoration, such as lung abscess or bronchiectasis, small amounts of the drug should be employed, (sodium pentobarbital gr. ½ and sodium amytal gr. 3 to 6), so as not to prevent interference with the cough reflex. Theoretically, the shorter acting barbiturates should not be administered in the presence of liver pathology, and sodium barbital and sodium phenobarbital should not be given where there is kidney impairment.

The barbiturates are contraindicated when large doses of morphine have been previously administered, because these drugs depress the respiratory center. The possibility of pulmonary edema or pneumonia from long periods of unconsciousness should be borne in mind.

THERAPEUTICS.

Barbital and its many derivatives, which are used for purposes of sedation and hypnosis, have superseded previous

drugs (opiates, chloral hydrate, sulphonal, trional, etc.), because of their relative margin of safety and also the belief at the time of their introduction that they were free from the power of addiction. Today, addiction to barbiturates for the sinister purpose of narcotic pleasure and enjoyment is a recognized evil. The old law of pharmacology, which teaches that efficiency varies directly with toxicity, has not been invalidated among the various barbital derivatives placed before the medical profession. Also, it is now generally conceded that all the barbiturates act alike qualitatively, differing only quantitatively, so that nothing is to be hoped for in the way of better and safer therapeutics from a barbiturate alleged to be effective in small dosage. The administration of barbiturate by mouth preliminary to the induction of general inhalation anesthesia has minimized the associated objectionable features, such as reduction of the preoperative anxiety of the patient, decreasing the required amount of inhalation anesthetic and diminishing the postoperative nausea. As a premedication for local anesthesia, it relieves the patient's apprehension, thus facilitating co-operation, and acts as a protection against cocain and procain poisoning. By animal experimentation, it has been shown that following preliminary administration of barbiturates in sufficient doses, the animal tolerates nearly seven times the toxic dose of cocain before convulsions occur.

In order for the clinician to consider the various drugs mentioned herein, it is necessary for him to understand the meaning of the various terms employed.

1. The *minimum lethal dose* (M.L.D.) is the smallest dose of any drug that will constantly cause a fatality. The pharmacologist *has determined* the M.L.D. by measuring the number of grams per kilo of body weight which will kill 50 per cent of a series of animals.

2. The *effective dose* is the amount of drug necessary, in the case of barbiturates, to induce narcosis.

3. The "*efficiency*" is measured by $\frac{\text{effective dose.}}{\text{M.L.D.}}$ It is the therapeutic coefficient; expresses the margin of safety and also determines the dosage.

4. The *presoporific time* (time of onset or latent period) differs in the various barbiturates and, therefore, a knowl-

edge of this factor is necessary for their proper administration.

The following comparative table of barbiturates shows the necessary data from which the otolaryngologist can choose the drug best suited for his needs. The order of numerical arrangement is the relative margin of safety and hypnotic efficiency from high to low. Column No. 1 represents the order of toxicity; that is, the relative margin of safety between the therapeutic dose and toxic dose. Column No. 2 shows the order of hypnotic efficiency; that is, the fraction of the *minimum lethal dose* that will produce hypnosis. This column also represents the presoporific time and the duration of hypnosis to complete recovery.

Name	1	2
Sodium barbital	7	1
Sodium phenobarbital	6	2
Sodium amytal	5	3
Dial	4	4
Alional	3	5
Sodium pentobarbital	2	6
Evipal	1	7

It will be seen at once that the toxicity of the drug varies inversely with its hypnotic efficiency, duration and time of onset. The barbiturates will now be discussed under method of administration.

INTRAVENOUS THERAPY.

The chief object of intravenous barbiturate therapy is for the purpose of producing anesthesia. The barbitals are insoluble organic acids which require large amounts of alkali for solution. On being injected into the blood stream they will not remain in solution (buffer action towards an alkali) and are, therefore, precipitated in a finely divided state. In all probability, the collapse resulting from intravenous injection may be due in no small part to the above phenomenon.

Although intravenous injection of barbiturates for anesthetic purposes are still used in many parts of the world, it has been found that oral administration is much safer.

The gastrointestinal and tissue absorption of barbiturates is definite and reliable, from which it follows that all the desired actions can be obtained by giving the drugs by mouth

and the collapse can be avoided; in fact, the sedative and hypnotic action are more satisfactorily controlled.

There are exceptions when prompt action is imperative for convulsions due to local or spinal anesthesia. From the evidence on hand, it appears that the barbiturate acid radical is the important factor in preventing cocain poisoning.

For routine work, it is well to have ready, for intravenous use, two ampules; one containing anhydrous sodium amytal (15 gr.), and the other containing enough sterile water to make a 10 per cent solution. For a very active hospital, 50 cc. ampules holding the following formula would probably be the best, but would have to be changed every three weeks because of the deterioration of the drug: Sodium barbital, 1 gm.; sodium chloride, 0.6 gm.; aqua distillate, 100 cc. From 50–75 cc. of this solution can be injected. The above is more stable than sodium amytal, and is also already in solution.

In cases where inhalation anesthesia is contraindicated, a barbiturate, called soluble evipal, can be administered intravenously, especially for those cases of short surgical procedures. It can also be used for cocain and procain toxicosis. It has a wide margin of safety, speed of induction, good relaxation and minimal disturbance of circulation. If the injection is made too rapidly, regardless of the quantity given, there is a fall in blood pressure along with diminished respiration. Death is usually respiratory in character. Delayed death is not demonstrable. If the patient does not succumb to a given dose within 15 minutes, he will survive. It is advisable that one with experience in its use should demonstrate the method of its application.

Evipal (sodium) at present is supplied in 1 gm. ampules and should be dissolved in 10 cc. of distilled water. The maximum dosage is 6 cc. of the 10 per cent solution for every 100 lbs. of body weight, or 0.06 cc. for every pound. The total should not exceed the 10 cc. at a single injection. The first 4 cc. should be injected at the rate of 1 cc. per 15 seconds and the remainder approximately 1 cc. in 10 seconds. It will be seen that good surgical anesthesia in most cases will occur after administration of 5 cc. (per body weight of 100 lbs.) of the above solution, and then the injection continued if the patient becomes semiconscious before the operation is completed.

ORAL ADMINISTRATION.

For the otolaryngologist, this is probably the least dangerous method of administration of the barbiturates. The comparatively slow rate of absorption is an additional beneficial factor, because a longer interval elapses before the drug becomes active.

Owing to this element of time, the margin of safety is also increased. It must also be remembered that this method of therapy only lessens, but does not eliminate the possibility of unpleasant complications. It is impossible to recover the portion of the drug which is active, but the potential advantage of the oral application is the removal, from the gastrointestinal system, of any barbiturate remaining unabsorbed. In figuring dosages for children over a year of age, Young's law should be borne in mind.

$$\text{Dose} = \frac{\text{Age}}{\text{Age} + 12}$$

For infants, Cowling's formula should be employed.

$$\text{Dose} = \frac{\text{Age}}{\text{Age} + 24}$$

It is always wiser at first to give a smaller amount, and if not effective, the dose should be increased. The following suggestions are offered for oral administration:

Sodium Barbital: This drug is used for slow and sustained hypnosis. It is relatively inefficient as premedication for inhalation anesthesia and prophylaxis against cocain and procain toxicosis. It can be used as an anticonvulsant in meningitis.

Presoporific time — one to four hours. Duration, 18 to 48 hours.

It should be preferably administered in capsules or powder; tablets tend to irritate the gastric mucosa.

℞ For sedation.
Barbital Solubile gr. xxxvi
Ft. Caps. xxiv
Sig. Cap. i three or four times a day.

℞ For hypnosis.
Barbitali Solubile ʒ ii
Ft. Caps. xv
Sig. Cap. i with hot beverage (milk or water) about one hour before retiring.

Sodium Phenobarbital: Sodium phenobarbital is used principally for sedation in insomnia, and to reduce the frequency for epileptic attacks. It is not generally used with analgesics or premedication for surgical procedures.

Presoporific time — one to three hours. Duration, 18 to 24 hours.

℞ For sedation.
Phenobarbital Sodii gr. xii
Ft. Caps. xxiv
Sig. Caps. i P.R.N.

℞ For hypnosis.
Phenobarbital Sodii gr. xxiv
Ft. Caps. xii
Sig Cap. i with hot beverage (milk or water) about one hour before retiring.

℞ For children or adults who may prefer liquid form.
Elix. Phenobarbital ʒ iii
Sig. ʒ i P.R.N.
Note: ʒ i of elixir contains gr. ¼ phenobarbital. For children one month old or over, make up prescription to contain only 1/16 of gr. of phenobarbital per dose.

Amytal Sodium: The action of this drug is slightly faster than barbital and also effective in smaller dosage. It is popular for premedication for inhalation anesthesia and as a prophylaxis for local anesthetic toxicosis.

Presoporific time — one-half to two hours. Duration, six to 10 hours.

℞ For sedation.
Amytal Sodii gr. ss
Ft. Cap. i
Disp. t.d. xxix
Sig. Cap. i t.i.d. p.c.

℞ For hypnosis.
Amytal Sodii gr. xxxvi
Ft. Caps. xii
Sig. Cap. i about one-half hour before retiring.

The premedication for inhalation anesthesia for adults, about a total of 9 gr. is used as follows: Three gr. the night before operation; and from 3 to 6 gr. one hour prior to operation. Morphine, gr. 1/6, and atropine, gr. 1/150, can be used, but it must be borne in mind that the former also depresses the respiratory center.

In the nervous or apprehensive patient, 3 gr. the night preceding operation, 3 gr. three hours before operation, then

6 gr. two hours prior to operation should be given. In minor operations, if the patient is to be ambulatory, the dose in all should not exceed 6 gr.

It is suggested that for children under 10 years of age requiring preanesthetic medication, from 1 to 3 gr. of sodium amytal be given one hour before operation and from 1 to 6 gr. in children over 10 years of age.

In preparation for local or regional anesthesia, the dose is from 3 to 6 gr. one hour before operation. For those who prefer amytal in liquid form, the following is used:

℞
Elix. Amytali ʒ iii
Sig. 3 i P.R.N.
Note: 3 i of elixir contains gr. ¼ of amytal.

Dial: This drug is a barbiturate with a therapeutic action similar to barbital, but is active in a relatively smaller dose. Its indications are, therefore, essentially the same as those for barbital.

Presoporific time — two to six hours. Duration, 10 to 20 hours.

℞ For sedation.
Dial gr. ss
Ft. Cap. i
Disp. t.d. xxiv
Sig. Cap. i 3—4 t.d.

℞ For hypnosis.
Dial gr. iii
Ft. Cap. i
Sig. Cap. i with hot beverage (milk or water) about one hour before retiring.

Allonal: This is a combination of a barbiturate and (amidopyrine) analgesic 1:1-2/3 and is useful in sedation and insomnia that is accompanied by pain. It should be remembered that amidopyrine and its derivatives are believed to cause agranulocytic angina in certain cases.

Presoporific time — two hours. Duration, five to 10 hours.

℞ For sedation.
Tabellae Allonal aa gr. ii 2/3
Disp. t.d. xii
Sig. Tab. i t.i.d.

℞ For hypnosis.
Tabellae Allonal xii
Sig. Tab. ii at bedtime.
In severe cases, Tab. iii may be ordered.

Pentobarbital Sodium: It is useful where a hypnotic of short presoporific time and diminished duration is desired. It is also useful for premedication in surgical procedures. For medical indications, such as nervousness and convulsions, another barbiturate with relatively prolonged effect is usually required. It is also useful for premedication in surgical procedures.

Presoporific time — one-half to one hour. Duration, three to six hours.

℞ For sedation.
Pentobarbital Sodii gr. vi
Ft. Caps. xii
Sig. Cap. i three or four times a day.

℞ For hypnosis.
Pentobarbital Sodii gr. xviii
Ft. Caps. xii
Sig. Cap. i about an hour before retiring.

As a premedication in inhalation and local anesthesia, 3 gr. divided in two doses, 1½ gr. the night before operation, and the other 1½ gr. about an hour prior to operation should be given. It should be used with caution for this purpose. For children, ½ to 1½ gr. one hour before operation is all that is needed.

Evipal (nonwater soluble): This drug is especially useful when a powerful, rapidly acting hypnotic of short duration is desired. The barbiturate is usually used to induce a hypnosis for insomnia and will be followed by natural sleep.

Presoporific time — one-half to one hour. Duration, two hours.

℞ For hypnosis.
Evipal gr. iv
Ft. Cap. i
Disp. t.d. x
Sig. Cap. i at bedtime.

This product is contraindicated in patients with diseased conditions of the liver.

RECTAL ADMINISTRATION.

In those patients who have disturbances in deglutition, these barbiturates can be administered per rectum, either by perforating both ends of the capsule and inserting as a suppository or dissolving the drug in a small quantity of water and given as a retention enema.

DISCUSSION.

In this symposium for the otolaryngologist, the relative value of seven barbiturates has been compared, and judging from the frequency of repetition in experimental data, it has been shown that they will serve every clinical purpose. The wide margin of presoporific time and duration shows that some individuals are more susceptible to the action of the drug and, therefore, the clinician should be especially careful in their application to the weak and emaciated. The use of intravenous therapy has been discouraged, except in special instances, because of the rapidity of the effect of the drug and the possibility of unpleasant, if not dangerous, reflex actions. Pharmacologists have shown that oral administration will serve every clinical purpose except one of emergency.

In closing, I wish to thank Prof. Maurice L. Tainter, M.D., and Aaron Lichtin, Ph.G., for their many helpful suggestions; also my preceptor, Prof. George M. Coates, M.D., to whom this paper is dedicated.

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1721 Pine Street.

A BENZEDRINE VAPORIZER FOR SHRINKING THE MUCOUS MEMBRANE OF THE NOSE AND EUSTACHIAN TUBE.

DR. SIDNEY ISRAEL, Houston, Tex.

For the purpose of shrinking the mucous membrane of the nose and Eustachian tubes, benzyl methyl carbinamine, otherwise known as benzedrine, is most valuable. It is a volatile vasoconstrictor. Clinically, it has many advantages over ephedrine solution. Its action is prompt, free from disagreeableness, and produces a maximum and reasonable prolonged shrinking effect on the mucous membrane.

For the past two years I have been using a container or vaporizer for the purpose of affording a convenient means of directly applying the benzedrine vapor to the mucous membrane of the nose and Eustachian tubes with the aid of compressed air or Politzer bag. The proximal end of the vaporizer may be opened by unscrewing the knurled flange for the purpose of loading the vaporizer with the benzedrine impregnated cotton cartridge. It is then closed by means of screwing the flange end in place, and is ready for use at all times and does not have to be refilled over a period of months, or until the impregnated cartridge has been exhausted.

The tapered or distal end of the vaporizer or container fits any size Eustachian catheter, also the new model DeVilbiss nasal tip. The proximal end of the vaporizer is so made that either a compressed air cut-off or Politzer bag tip is readily adaptable. Therefore, this container serves a two-fold purpose. With the DeVilbiss nasal tip attached to the distal end of the vaporizer and inserted into the nostril, and the compressed air attached to the proximal end, the benzedrine is vaporized and directed into the nose, which causes a prompt and convenient shrinkage of the mucous membrane. With the nasal tip removed and the tapered distal end inserted into the Eustachian catheter, the benzedrine vapor is directed into the Eustachian tube through the catheter by means of a Politzer

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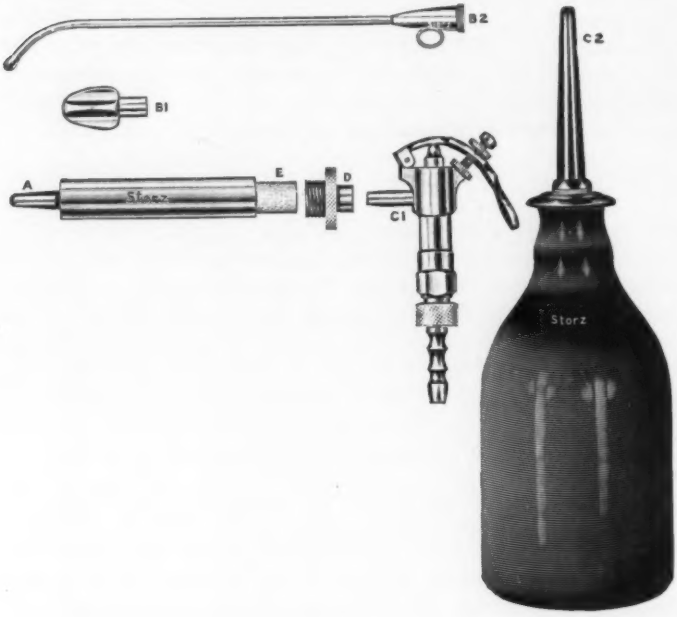
bag or regulated controlled compressed air and cut-off, whichever the choice may be, in order to contract the mucous membrane of the Eustachian tube.

The vaporizer is made of one piece, except as described above, and when once loaded, no preparation is necessary between treatments. The purpose of the knurled flange is to give support for the fingers when the distal end is inserted into the nose or Eustachian catheter. Its simplicity and effec-



tiveness for the purpose for which it is employed is everything one could desire.

The vaporizer is made by the Storz Instrument Co., of St. Louis, Mo., and the benzedrine cartridge is inexpensive and readily obtainable through either a local pharmacy or the manufacturers, Smith, Kline & French Laboratories, Philadelphia.

2010 Niels Esperson Building.

IN MEMORIAM

DR. EDWARD BRADFORD DENCH

1864-1936.

After a protracted illness that incapacitated him for two years, Dr. Edward Bradford Dench, outstanding American otologist of New York City, died of cerebral hemorrhage, Feb. 21.

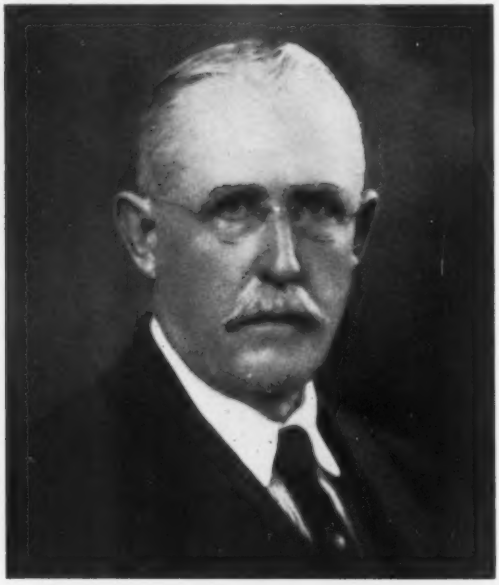
Dr. Dench was born at Leedsville, N. Y., the son of Josiah Bradford and Frances Lester Dench. He was a descendant of Gov. Bradford, of the Plymouth Colony, and of Capt. Robert Dench, who migrated from England to Boston in 1715.

In 1883, he received his Ph.B. at Yale University, and in 1885 his M.D. Degree from the College of Physicians and Surgeons of New York. He was Professor of Otology at University and Bellevue Medical College, 1898-1930, at which time he became Professor Emeritus; Senior of the active staff of New York Eye and Ear Infirmary; Consultant in Otology at St. Luke's Hospital and New York Orthopedic Dispensary and Hospital.

An active factor in the organization of the American Laryngological, Rhinological and Otological Society, he was its first President, in 1896, and in 1934 was made Honorary President, the only Fellow of this organization to receive this special honor. For many years he was an active member of the American Otological Society and was honored with the presidency of this Society in 1911. He was President of the Section of Otology of the New York Academy of Medicine, 1904-08; President of the New York Clinical Society in 1909, and active in numerous other medical organizations.

In the World War, Dr. Dench held the rank of Major in the Army Medical Corps and was active at Camp Upton during the enlistment period. He held membership in the Yale and Union Clubs and the Sons of the American Revolution.

His contributions to the programs of the various special societies of which he was a member were frequent and important and always gave evidence of his keen observations as a brilliant technician and clinician. Perhaps no American otologist has enriched the clinical field of his specialty to a more effective degree than he. His textbook on Otology has been recognized for many years as one of the most authori-



tative and comprehensive in the English language. His vast clinical experience, fertile brain and facile form of address qualified him to an unusual degree as a clinical teacher in otology and in this capacity he also greatly enriched the otological profession.

He was a man of determined character, fearless in the expression of his scientific convictions and always respected

for his viewpoints; his personal refinement and fund of general information made him a much desired member of any scientific or social group.

In 1888, he was married to Miss Marie Antoinette Hunt, who, with a daughter, Mrs. J. Russell Hawks, of Detroit, survives him.

In the death of Dr. Dench, American otology has lost one of its most valued pioneers and supporters; one who stood for its highest ideals and who enriched its clinical literature beyond the usual measure.

To his family and intimates we express our sincere sympathy.

M. A. G.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Dec. 13, 1935.

(Continued from March Issue.)

DR. HORACE NEWHART gave some remarks on acoustics as related to otology.

Dr. Horace Newhart, introductory to the presentation of two sound films, "Sound Waves and Their Sources" and "Fundamentals of Acoustics," furnished by the Department of Visual Education, made the statement that a lack of familiarity with the fundamentals of acoustics has materially retarded otological progress.

In practice the fact has largely been overlooked that no accurate test of hearing can be made in the presence of a noise. He pointed out the importance of detecting relatively slight degrees of hearing impairment as indicative of the presence of beginning pathology of the ear, both in the young, when most frequently the capacity to hear lower tones is affected, and in adults, when loss of the acuity of hearing for the high tones is frequently overlooked until well advanced.

The methods which have been employed in the past have been too crude to disclose the slight losses, and we have failed to exclude sufficiently the ordinary noises which mask the test sounds of slight intensity.

The remedy consists in excluding the noises which ordinarily mask the sounds employed in making the tests and in using a satisfactory audiometer. The telephone receiver of the audiometer itself excludes some of the extraneous noises which interfere with accuracy.

The prevailing noise level found in a downtown office or school building for practical purposes can be reduced by using an acoustically-treated booth, so as largely to exclude masking or distracting noises. This can be constructed at a moderate cost.

In response to a rapidly growing need among otologists and distributors of hearing devices, which has been stimulated by an increased demand for modern hearing devices, many new audiometers are now in process of development. Some of them, when tested in the acoustic laboratory, as is now being done at the University of Minnesota under the direction of Dr. Henry E. Hartig, are still in the developmental stage and, in their performance, leave much to be desired. They already offer several improvements over older models, as greater range of operation, increased portability, greater ease of operation, and lower cost and upkeep.

An audiometer to meet the minimum requirements of an otologist must be capable of producing tones of a high degree of purity over a range of from 64 d.v. to 8192 d.v. at intervals of not less than one octave and having an operating range from the threshold of normal hearing up to practically the threshold of feeling. It should be free from internal noises; have a satisfactory, standardized bone conduction receiver; should be readily portable; of reliable construction, and be sold at a moderate price.

Such an instrument will be a most useful part of the equipment of every physician practicing otology and will save him the embarrassment of not being as well equipped as the "audiometrist" who is selling hearing devices.

DR. FRANK L. BRYANT, (by invitation) reported a group of 28 external fronto-ethmo-sphenoidectomies done in the Department of Oto-Rhinology at the University of Minnesota.

Abstract: The technic used was that suggested by Lynch, Sewall and Ferris Smith. Patients who showed a third or fourth degree chronic sinus infection were operated by this method. Both nonallergic and allergic types were considered.

a. In the non-allergic group the chief complaints in order of frequency were: 1. Purulent discharge almost continuous. 2. Nasal obstruction, usually complete; not influenced by intranasal medication, and those operated upon by intranasal methods were reoccurring. 3. Headache, a variable complaint, usually occurred during and following an acute exacerbation.

b. In the allergic group asthma was the outstanding complaint, with the afore-mentioned other occurring in the same order.

No patient who had a temperature of over 99.6° was operated. All operations were performed under local anesthesia.

Pathologic Findings: Surgery as a rule showed more extensive changes than clinical and X-ray findings would indicate. Histologic studies showed leukocytic infiltration and marked fibrosis.

Postoperative: Patients were instructed to refrain from blowing the nose. This minimized orbital edema; they were not allowed to used their eyes for reading for at least 20 days. No diplopia or epiphora were encountered in those who followed this order. Postoperative discomfort was usually relieved by codein, ½ gr. Only one patient showed a postoperative febrile reaction of 101°. Others had temperatures ranging from normal to 100.3°. Hospitalization period averaged from five to six days.

Results: There were no deaths. There were no complications. Only one patient had a slight epistaxis on the seventh day. Scar from incision was neglible — most of them were not visible after six weeks.

Results from chief complaints: All purulent discharge and nasal obstructions were markedly improved to clinically cured. At least seven of those who had headache or neuralgia were much improved. One who previously required morphin is now free of all discomfort. In the allergic group two have had no asthma since the last surgical procedure. One has asthma after acute rhinitis. The others have antrum surgery to be done.

DISCUSSION.

DR. H. L. WILLIAMS stated this paper is so clearly presented there is little to say in additional discussion of this type of operation. The techniques are somewhat variable but all follow the operation of Jansen. Dr. Williams felt the important thing is the selection of cases. He felt it is not applicable to early sinus disease, but where there is sclerosis of the bone and in cases which have had repeated operations, this operation is best because in that type of case the intranasal operation is virtually impossible.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Jan. 10, 1936.

DR. J. J. HOCHFILZER reported the following case of osteomyelitis of the maxilla.

The patient presented this evening, a girl, age 20 years, who was seen for the first time in the out-patient department of Ancker Hospital on March 19, 1935. Her chief complaint was a purulent nasal discharge, nearly constant for the last three years; a nasal growth (probably a polyp) had been previously removed; and had had several rheumatic attacks in her leg joints.

Examination revealed a marked deflection of the septum, a hypertrophic right lower turbinate and bilateral purulent nasal discharge, series I, more pronounced on the right side. X-ray findings were: "Antra, cloudy, grade 4; ethmoids, cloudy, grade 3." A number of antrum lavages were done in the next two months, which always produced a purulent flaky secretion of foul odor. The condition in the left antrum was similar in the beginning, but improved considerably.

On May 6 a submucous resection of the nasal septum was performed; recovery was uneventful, but no improvement of the condition of the right antrum took place. On June 1 a window resection was done; in the description of the operation it is stated that the anterior third of the right lower turbinate was severed from its attachment to the maxilla by incision; the posterior portion infracted to get better access to the lower meatus; the window resection was performed with rasp and biting forceps—the antrum was found filled with pus and polyps; some of it was removed and the floor of the antrum curetted.

The next morning the temperature rose to 103° and a marked swelling was noticed over the region of the right maxilla, especially the frontal process and inner canthus of the eye. An elevated temperature persisted for about two weeks; hot packs were applied and the swelling subsided considerably, but that area remained very tender.

On June 25 an incision similar to the Caldwell-Luc operation was made over the right canine fossa, the periosteum was elevated toward the inner canthus and inferior orbital ridge, but no pus obtained, and an iodoform drain was inserted. The temperature gradually came down to normal, but there was still a markedly infiltrated, hard, tender area over the right frontal process. The patient was discharged on July 2 and the antrum condition was treated by repeated suction and improved considerably.

For the last six months the patient's condition can be summarized by stating that she had persistent pains and swelling in the region of the frontal process and a moderate purulent nasal discharge. When the writer saw the patient three weeks ago, palpation of that area revealed a sharp pointed bony structure in that region, very painful on pressure, skin adherent and retracted at that point. The impression was that one was dealing with an osteomyelitis of the frontal process of the maxilla and bone sequestration. The X-ray report of Dec. 31, 1935, is as follows: "Re-ray shows what appears to be a localized area of destruction medially in the infraorbital portion of the right maxilla, which may represent localized osteomyelitis. This is approaching the infra-orbital nerve canal."

Dr. Hochfilzer said he did not want to go into any lengthy discussion in regard to the pathogenesis of osteomyelitis of the jaws at the present time, as he intends to present a more extensive essay on this subject some time later on. Most osseous lesions of the jaws are due to the spontaneous extension of the infection along vascular channels into the jaw bones from an area of infection in close proximity to the bone. Extension of bone necrosis depends, as in other bones of the body, on the importance of the vessel or vessels occluded by the osteomyelitic process. The blood supply of the superior maxilla is very abundant, nearly all branches are derived from the trunk of the internal artery.

There is no doubt that the operative trauma at the time of the window resection is the causative factor; whether the incision or infraction of the turbinates or the curettement of the antrum cavity was responsible for it, is difficult to say; but the writer believes it was the incision of the anterior portion of the right lower turbinate, because that portion is attached to the so-called crista conchalis of the frontal process of the maxilla, and in this way the avenue for the spread of the infection to the bone was opened. In the writer's opinion the most advisable treatment would be to do a Caldwell-Luc antrum operation and, at the same time, attempt to reach the area of osteomyelitis and remove the sequester.

DISCUSSION.

Dr. W. H. Hanson said that to him this was a very interesting case, because he had had two deaths from osteomyelitis of this type. It pops up most unexpectedly in cases which may seem like a very mild case of sinus infection, and spreads like wildfire. One case had a tooth infection which subsequently developed to a sinus infection. The dentist pulled the tooth and Dr. Hanson had done a subturbinal window drainage without curetting the sinus or elevating the periosteum. The man died in three months of venous sinus thrombosis and meningitis. Dr. Hanson had also seen another case with his associate. He punctured the sinus and enlarged the opening with rasp. This man also died from osteomyelitis. When the next case came along, Dr. Hanson said they were rather desperate, so they resected the whole superior maxillary bone. In another case he removed the sequestrum at the thirteenth operation and this man is still alive.

Dr. Hanson felt there is a difference in the prognosis, depending on whether the osteomyelitis occurs as a result of a sinus infection or after an operation upon an infected sinus. If osteomyelitis occurs, following a sinus operation, the prognosis is extremely bad; the infection gets into the diploic space and then into the diploic veins, which lead into the venous sinuses, and before death occurs there is a sinus thrombosis and meningitis.

The fact that this case shown tonight has run along for about six months without such a vicious course seemed to Dr. Hanson to offer a better prognosis. The cases which he had, had formed no sequestrum.

Dr. H. L. Williams read a paper on "Primary Operation for Suppuration of the Petrous Pyramid."

Two cases were reported, which were designed to illustrate the symptomatology of petrositis as it appears in a classical example, but more especially to indicate that primary diagnosis of petrositis is possible, and that at the first operation the disease in the petrous pyramid, as well as the mastoiditis, could be taken care of. The literature was reviewed and an attempt made to touch only the outstanding contributions. The contribution of Baldenweck was particularly stressed.

DISCUSSION.

Dr. L. R. Boies said that Dr. Williams referred to the radical mastoid and he wondered whether he meant a complete mastoid or a radical mastoid?

Dr. V. J. Schwartz said he would like to mention briefly two matters in connection with this paper. Dr. Williams reviewed the literature very admir-

ably, but he neglected to mention what Dr. Schwartz considered a good piece of work, namely, that of Profant, and Geyman, of Santa Barbara, which was demonstrated at the A. M. A. meeting in Detroit four or five years ago. This work, and the exhibit which accompanied it, was particularly interesting from the standpoint of radiographic visualization of the petrous cells.

He also wished to ask Dr. Williams if he had any explanation in the first case as to why there was complete paralysis of the IIIrd nerve, but no paralysis of the VIth, which is much more commonly involved, when both are not affected.

DR. WILLIAMS, in closing, said he believed Dr. Boies was referring to a quotation from Baldenweck. Baldenweck advised a radical mastoidectomy, but at the Mayo Clinic they differ from Baldenweck in that they believe a radical mastoidectomy is rarely necessary.

In answer to Dr. Schwartz, Dr. Williams said he was familiar with the work of Dr. Profant and held it in great respect, but he had not attempted to make a complete review of the literature, which, due to its present voluminousness, would be extremely tiresome. but he had tried to touch only on the salient contributions. So far as the paralysis of the IIIrd nerve is concerned, Dr. Williams said that, according to Atkinson, this symptom is due to pressure of the IIIrd nerve against the carotid artery. He pointed out that it was not his intention to give the impression that all the symptoms mentioned by the various authors were necessarily present in any given case of petrositis and that, as a matter of fact, petrositis was occasionally practically symptomless. He said that he had a patient who had suddenly developed meningitis, although he had been free of symptoms for more than a year following an acute mastoiditis. At autopsy it was found that involvement of the petrous pyramid had occurred along the inferior line of cells.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of Dec. 18, 1935.

A Simple Mastoid Operation and Some Functional Results. Dr. Fred W. Graef.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JAMES G. DWYER: This is a very timely subject and I think Dr. Graef has covered the subject very thoroughly. We must discuss the question of the simple mastoid operation from two standpoints. In some cases the question of hearing is absolutely secondary to the saving of the patient's life. In other cases we can very definitely decide to operate for the preservation of hearing alone. In reference to the first type of case, just within the past three weeks I saw a case where they had to do a radical and go way forward into the tympanum and ear after it had run only three weeks. The boy had developed an extradural abscess in that time.

The question of trauma is very important. When I first began operating, we were curetting the granulations in the antrum. We have stopped that. Most of the trauma, I think, is due to a too thorough cleaning of the aditus. We have been taught that the cardinal principle is to clean out the antrum. In the past 15 years I have not bothered with the antrum at all, on the principle that the granulation tissue there is simply the productive type due to the organism, and once you establish drainage these granulations disappear. Another important point is that we get dislocation of the incus most often in working in the aditus. We perhaps dislocate the incus a little and get what corresponds to a lame joint, an arthritis, with a form of deafness as the result.

The fundamental question to me, one which I have never been able to understand, is the contrary results we get in hearing with the pathological findings in the middle ear afterward. I remember a doctor's son, now age 17 years, on whom I operated 15 years ago at the age of 15 months for tuberculosis of the mastoid. He had tuberculosis of both mastoids from certified milk. I reported this case. We were able to get the bovine tubercle bacillus which came from one infected cow. I did one mastoid on account of the urgency of the acute symptoms. You could lift out practically the whole mastoid process as one ulcerated mass. The other one was allowed to go on and clear up by itself. The boy has no ear drum and still no one knows that he is deaf. Another case bearing on that same point is one which I followed three years ago this Christmas for seven weeks. The patient refused operation. There was a perfectly tremendous perforation of the drum. It cleared up and reformed with what we call a tissue paper scar. She has less than 2 per cent loss of hearing. Another case that was a lesson to me was one which Dr. Haskin followed for six months. The patient was diabetic and refused operation. There was a subperiosteal swelling, which finally cleared up with normal hearing. That was before we had the audiometer.

One point which we ought to stress and keep in mind is the question of the organism. You have all seen cases where practically two-thirds of the drum has been destroyed in a few days, for instance, due to the toxic action — let us call it that — of the streptococcus or pneumococcus. Other cases run for weeks and weeks and the hearing seems to be just as good as if we had operated in the first week.

Another point is the question of our conceptions of how long an infection has lasted. I think we must review our conceptions because I recall a girl on whom I did a double mastoid about three years ago, after a consultation on the seventh day, on account of the urgency of the symptoms and prodromal symptoms of meningitis. These mastoids were absolutely guttered out. I like to watch these cases and not go in until the mastoid becomes locally immunized, but I have come to the conclusion that the history in many of these cases is very misleading and a lot of them may have been 10 days or longer going through lymphatic tissue, and the mastoid symptoms may be due to infection going forward, rather than from the middle ear backward.

We can hardly draw conclusions as far as duration, etc., are concerned, and we must look at the subject from two standpoints: first, as to the sacrifice of hearing, if it is a question of the patient's life; and, secondly, if the acute symptoms have disappeared and the hearing is rapidly deteriorating, we are justified in operating in that case for the preservation of hearing alone.

DR. EDMUND PRINCE FOWLER, SR.: I think it is high time that we really did start to do this work which has been brought out in Dr. Graef's paper. Very little testing of the hearing has been done in these acute cases, either afterward or before the mastoid operation. If the cases reported had been tested before the mastoid operation, when the child was apparently normal, I believe it would have been found that there was very much less change in loss of hearing afterward. A great many of these acute mastoid cases have had a prior attack, even though we cannot get a history of it. This is shown by autopsy findings. I do not believe that the mastoid operation per se, properly done, as most of us do it, is the cause of the loss of hearing. The loss of hearing is caused by the adhesions that follow in the wake of middle ear lesions because of too long waiting for efficient drainage.

There is a critical time for the mastoid operation in the acute cases, but I think we all agree that we should wait until a certain resistance has been set up before we operate. Every case must be handled as an entity. You cannot lay down standards or rules for operation on a mastoid in the first week or the second week, or the third or fourth weeks. It depends on the clinical history and on your observations in that particular case. Many cases with middle ear suppuration have been going on for weeks before the ear symptoms are discovered. If you use the nasopharyngoscope you will often see pus coming from the Eustachian tube, while in the ear there may be very little trouble visible. Such cases will often subsequently get up a mastoid. The main point I wish to bring out is that, in my opinion the mastoid operation per se does not bring about loss of hearing. In fact, it is one of the greatest aids for the preservation of hearing, if properly timed and executed.

The Interpretation of Clinical Tests of Hearing Acuity. Dr. Stacy R. Guild.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. EDMUND PRINCE FOWLER, SR.: One cannot help but agree with Dr. Guild on all these facts or observations, and one is apt to go along with him in his reasoning, but I suppose it is the duty of a discusser to try and bring out as much as he can of the opposite side in rebuttal.

In the first place, facts are not always what they seem, as Dr. Guild would be the first to admit. These specimens are all autopsy specimens. These cases were examined at a considerable time before death and before the specimens were made. Changes take place in the interim. Nevertheless, this is the best we can do. All autopsy work has this fault. It is reasonable to suppose that even though some tissues have changed since they were examined, there remain a certain percentage which have not changed much and, therefore, can be considered as representing the antemortem conditions.

Some of the graphs he has shown just do not make sense. In the first place, if masking is too loud it destroys the accuracy of measurements. This is one of the faults of the Barany apparatus. Another objection is that unless you use two Barany apparatuses, to be sure both ears are cut out, you are not sure you are cutting out the masked ear when testing the other.

As for the age of the patient, it is a fact that as we get older our hearing on the average goes down, both by air and by bone conduction. The charts of Bunce and Rayford show this, and those I showed some years ago also show it in cases with cardiovascular disease. Most old people have arteriosclerosis, and they show therefore practically the same graphs as Bunce and Rayford showed, but in these same cases there were 5 per cent who did not show any loss even up to 70 years of age, and 10 per cent who did not show it with bone conduction, even up to 70 years of age. Therefore, it was not the age that did it. It was something else. Nevertheless, as a general statement, it is perfectly true that with age our hearing does go down, particularly for the higher tones. Is there any normal for any age? When we speak of normals we are speaking of the average for the cases we have examined. Even our normal line is 10 decibels below the very best and 10 decibels above the worst of the supposedly normal ears, a spread of 20 decibels, or 20 units. Dr. Guild states very correctly that in a noisy place we do not consider a 20 decibel loss a real loss. This is often true. Even in this room, it may seem to be quiet, but there is enough noise here to mask the hearing tests.

Dr. Guild has shown cases where there appears to be nothing to account for the deafness, and other cases where the most remarkable lesions failed to cause deafness. I think this is a very important thing to show. You cannot show it too often. It is not otosclerosis or adhesions, or a blood clot or a collection of mucus or thickening or any of the different things that occur in the middle ear that matters, but the particular position they happen to be in, or happen to affect, principally the stapes and the round window. If the round window is blocked, I cannot conceive how there can be very much hearing, either by bone conduction or air conduction, because there is no escape or give and take for the pressures in the labyrinth. If the oval window is blocked you can still get perfect bone conduction, even better than what we call average normal, but of course air conduction is down. One reason why these high tones may be affected more as we get older is that the longer our span of life, the more we are subjected to inflammatory episodes in the ear. It is very rare indeed to find an ear without signs of such an episode, and every one of these affects not only the mucous membrane of the middle ear but very often extends beyond it and affects the circulation in the base of the cochlea, and particularly the circulation to the ganglion cells. Therefore, the older we get, the more episodes and the more apt we are to have a diminution of our hearing for the higher notes, rather than the lower notes. This is just an hypothesis, but I am not one who frowns on suppositions and hypotheses. I think we must set them up to point the road for future progress. Let us not stop romancing and having hypotheses and guessing, because we will not get anywhere unless we do.

When you and I talk about nerve deafness we are not saying an awful lot. We are saying about what we said in the old days when we stated there was fever. When we say a man has nerve deafness we are saying just about as much. We are not saying where it is, in what part of the membranes, the ganglion cells, the nuclei or the cortex, it is in, or whether it is in the connections in the brain about which we know so little. This is one of the fields for research hardly tapped. We commit a like error when we say syphilitic deafness. If we find a case with high tone deafness and a 3+ or 4+ Wassermann, we call it a syphilitic deafness, which does not follow at all. Very often you find perfect ears in cases dying of syphilis.

When we say that nothing was heard (we all use that term, and, as Dr. Guild said of some of the charts, that this certain fork or frequency was not heard, and so on), we mean that we simply did not make it loud

enough for the patient to hear. That is all. Even in these extremely high tone losses, where the tuning fork is not loud enough to be heard, where the 16,000 or even the 8,000 audiometer tones cannot be heard, often the patient can hear the Galton whistle. They hear this because it is very loud. So we have to take all such data with limitations.

Just a word in regard to localization: I think opinion on localization is changing to this extent, that no one thinks now that this means localization at a point in the cochlea. We mean an area or spread. You cannot get around the fact that everything has a localization for vibrations, so that the cochlea is going to do the same thing. This may have no bearing on the hearing. Nevertheless, there is localization. There has to be, and the cochlea seems to be constructed beautifully for localization. However, it is not so easy to prove the exact physiological operation.

Masking is a subject which I am very glad Dr. Guild brought up. I think we have all been remiss in neglecting masking. We think that because two ears happen to test alike that they are hearing discretely and are not at all being influenced by the other ear. That is, of course, not true. We even think that if the one is 40 to 50 units down from the other ear that we are not getting a shadow picture.

I can only explain the case Dr. Guild showed where the two were so close together and he got a shadow picture, by saying that perhaps his noise apparatus was making too loud a noise. That would explain it. Otherwise, it seems almost inexplicable. I have never run across such good hearing with such a nearness of the two audiograms with total deafness in one ear.

Regarding the fissures, I would like to know if Dr. Guild has tested any of these cases after a radical operation; after taking out all the bone in the site he mentions, I predict the bone conduction will be found excellent, even what we call above normal. Here is the point. In doing a radical, we take out most of the bone which Dr. Guild mentioned, and yet they hear well with the tuning fork anywhere on the head. That is just one point. Dr. Guild, do not think I do not sympathize with you. Yours is a wonderful theory; but I bring up the other side. Of course, we do find these cracks in the bone more in old people, and they are practically filled with fluid because they are filled up with a fibrous connective tissue which can be considered as fluid for all intents and purposes. It is not a vacuum nor is it air. I cannot see any reason why the sound cannot go around these cracks. What is the difference between the low tone and the high tone conduction in these cases? Are they affected differently? If this theory is true unless there is a lesion to account for air conduction loss, antemortem, you should get, at least occasionally, no diminished air conduction. Such a result I have never seen.

Dr. Frederick A. Hemsath: I would like to express my great pleasure in having the opportunity of seeing some of the work from Dr. Crowe's laboratory in Baltimore. Not being in a position to do hearing tests, I do not feel qualified to speak of them, but I do admire the beautiful sections that Dr. Guild has shown and I must say I agree with him in his interpretation of the pathology. There is nothing in which he made the error of taking artefacts for a pathological condition. As I listened to his paper I can only say I felt again the foresight and wisdom of Dr. Crowe in organizing his department for research as well as for clinical study at the Johns Hopkins. I recall the remark of the dean of one of our prominent medical schools in addressing a small group of men. He said that there was only one department of oto-laryngology in the medical schools of this country which was modern, and he was not the dean from Johns Hopkins.

Dr. Edmund Prince Fowler, Jr.: There are a number of small points which I think might be emphasized in Dr. Guild's paper. It is interesting to see the cases which had no loss of hearing and yet pathological lesions were present. My father and I have shown that in otosclerosis there is often thick-

ening of the lining mucosa of the middle ear in one place or another, suggesting that there must have been a preceding inflammatory process. The latter is a possible etiological factor in the production of otosclerosis.

Dr. Guild also brings out that it is possible to get a hearing loss without an apparent lesion histologically. Last April at the meeting of the Society for Experimental Biology and Medicine I made a preliminary report of some experiments on cats, in which sodium chloride was placed on the round window. A marked reduction of the Wever and Bray effect was found; that is, the electrical pickup from the cochlea was much decreased and yet the placing on the round window of nonelectrolytes produced no change in the response. Sections of the temporal bones of the animal often showed very slight histological changes. How much this has to do with human hearing I do not know, but it is possible that the lack of histological evidence of loss of hearing in the one-quarter of the cases mentioned by Dr. Guild may be explained by some electrolytic poisoning which is only apparent physiologically.

I find the cases of acoustic neuromas mentioned by Dr. Guild particularly interesting. I have recently been reading about this type of lesion and we have four cases in the laboratory at Columbia. His cases showed no symptoms. All of those we have showed different symptoms. I think this is important because if you read the books of Fisher and Cushing you find that they have laid down quite definite dictums about the "characteristic symptoms" of acoustic neuromas. Our first case was a very small tumor like those shown by Dr. Guild. The only symptom was tinnitus. The patient was an old man, who possibly might have had tinnitus from some other cause. At any rate, this was the only symptom that might have been due to acoustic neuroma. The second case complained of deafness. She had no tinnitus, no nystagmus and no vertigo. On section a neuroma was found entirely within the auditory meatus and it had completely destroyed the end organ of both cochlear and vestibular labyrinth. The other two cases were cerebellopontine angle tumors. One was quite large and showed a vestibular loss and no cochlear loss except in the high notes. The other showed complete vestibular, complete cochlear loss and a little facial weakness. I think it is very important to realize that acoustic neuromas may cause no symptoms whatsoever, or, on the other hand, may cause any one of a series of symptoms, either vestibular, cochlear or facial.

In speaking of bone conduction testing, Dr. Guild mentioned that the 512 tuning fork was used exclusively. I think it was unfortunate that his bone conduction readings were not made with the same instruments as his air conduction readings (an audiometer), but there is no question that the use of the 512 fork is the next best procedure. Peculiarly enough, most hospital examining trays contain only a 128 fork, and this is used routinely for bone conduction tests. This is perfectly absurd and I think it is one of the reasons why bone conduction tests are often apparently so peculiar. Anyone can *feel* a 128 fork, even on his tibia (if he hasn't got tabes). The difference between feeling and hearing is so slight that it simply cannot be differentiated at 128 double vibrations. The 256 fork is more difficult to feel and can be used, but of course the 512 fork is the proper one and I think it is about time that hospitals where neurological examinations are done supply a 512 fork for routine bone conduction tests if they have not the bone conduction apparatus of an audiometer available.

DR. JOHN GUTTMAN: The examination of the function of the ear is one of the most difficult undertakings which an otologist has, and there are two methods of going about it. One is the method which the speaker of the evening employed, the clinical and histological-anatomical method. This is done by examining the patient clinically and then correlating the findings with the histology. I must add here that if anyone is competent to do this work, it is Dr. Guild, who is probably one of the best anatomists of the ear in the country. However, there is one shortcoming in this method, and that is that at postmortem changes are produced causing artefacts in these very delicate

structures in the ear, and it is very hard to say whether these changes are due to the ear lesion or are artificially produced. The other method of examining the function of the ear is the so-called animal experimentation method. Here we produce these lesions in the animal and see what we can find afterward, what changes of functon are produced by our lesions. The only shortcoming of this method, again, is that we cannot tell when the animal hears. We do not know that. It interferes with the examination. There is, however, a method used, especially in the past few years, wherein it is found that stimulation of the ear, as by a sound, is accompanied by an electrical change, a so-called action current or electrical potential. We can see this electrical potential objectively, by using the voltmeter, the galvanometer, the telephone receiver and a few other methods. This helps a great deal in using the animal experimentation method.

I have used this animal experimentation method in the past five years on hundreds of animals, mostly cats, some rabbits, some rats and some monkeys. I cannot go into the details here, but I want to make one statement: I found this electrical change after using stimulation of the ear by sound, even after resection of the nerve. This is something which is difficult to explain. There are some who try to explain this phenomenon by saying that although the nerve is severed there are still filaments between the cut end of the nerve and the cochlea, and so on, and that these are the elements which give this action current observed in the experiment. Dr. Barrera and myself degenerated this spiral ganglion in several cases and found that this phenomenon, this electrical disturbance, was present even after the degeneration of the spiral ganglion. That is something which makes us think.

Dr. Guild in his modesty said he would recite only facts, not make interpretations, and Dr. Fowler was delightful in saying he was addicted to romancing and using hypotheses, because, without them, we can get nowhere, and we can always change them later. He claims that Dr. Guild's facts are not so. If they are not so, they are not facts, so that he was mistaken in his facts. I think this discussion will help us to look further into this matter and study it further.

MAJ. WALTER FRANZ VON ZELINSKI: I thank you very kindly for your invitation to speak. I have nothing whatever to add to this very excellent paper. It has opened up some avenues of speculation for me. In my time I have tested the hearing of some hundred thousand men, and many things have come up to cause me to speculate on what I have found and have caused me to experiment in my own crude way. I have been trying out, when the opportunity offered, the experiment of testing the hearing with two forks of different pitch simultaneously to see if I could learn anything by such procedure, but I am not prepared to say anything about the subject now.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Fifty-Eighth Annual Congress of the American Laryngological Association will meet at the Hotel Book-Cadillac in Detroit, on May 25, 26 and 27, in conjunction with the American Bronchoscopic Society on May 27, and the American Otological Society on May 28 and 29. The Council will meet on Sunday evening, May 24, at Dr. Shurly's log cabin on Grosse Ile. The opening session of the Congress will be at 10 o'clock on Monday morning. The annual banquet will be held on Tuesday evening, May 26. President Frank Cody, of Wayne University, will be the guest speaker and special entertainment will be provided.

The Council has recommended the following for election: Honorary Fellowship, Mr. Herbert Tilley, of London, England; Corresponding Fellowship, Dr. John G. Fraser, of Edinburgh, Scotland; for Emeritus Fellowship, Dr. Joseph L. Goodale, Boston, Mass.; Active Fellowship reinstatement, Dr. John F. Barnhill.

President and Mrs. Shurly are in charge of the social program, and have invited the members and their wives to an informal reception and smoker on Monday evening in the Abbey of the Detroit Athletic Club. Dr. and Mrs. Furstenberg have invited the members and their wives to dinner and entertainment following the Wednesday afternoon program.

Reservations at the Book-Cadillac Hotel should be obtained at once.

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